ELITE OBS/GYN

BASED ON LATEST STANDARD TEXTBOOKS

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More than just notes...

Obstetrics & Gynaecology

We hope you enjoy using these notes

They have been hand-crafted with an obsessive attention to details, with aim of capturing the course's content in a way that's right for you.

Thank You

For the Students
from the Students

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HOW TO USE THIS NOTE

Edition II

- High Yield Concepts
- Referred from the latest Standard textbook.
- Direct reference from National faculty lectures.

CONGRATULATIONS

You now posses the book that will guide Medical PG Aspirants to score the best rank in PGMEE. With appropriate care, the binding should last the useful life of the book. Keep in mind that putting excessive flattening pressure on any binding will accelerate its failure. If you purchased a book that you believe is defective, please **immediately** return it to place of purchase.

FOR BETTER RESULTS

Start Early :

Use this notes as early as possible while learning the basic medical science. The first semester of your first year is not too early! Devise a study plan and make an early decision on resources to use and do not shift on your resources. Its always better to keep things simple and stick to one resource rather than confusing yourself with multiple sources.

Consider *ELITE NOTES* as your annotation hub :

Annotate material from your primary resource, such as lecture class or videos, into your notes. We have already referred and incorporated almost all information from the available sources. This will keep all the high-yield information you need in one place.

BOX ANNOTATION :

We have introduced concept of Box annotation

1. **RED BOX :** Includes the most repeated topic along with the next potential MCQ that we believe will be tested in future exam
2. **BLUE BOX :** AIIMS/ NEET PG new pattern question
3. **PURPLE BOX :** Recent NEET PG recalls

Prime Your Memory :

The visual representation of concepts incorporated into the notes serve as a useful way of retaining key associations and keeping high-yield facts fresh in your memory just prior to exams.

CONTRIBUTE TO ELITE NOTES :

No resource is error free and we do not guarantee the notes are free of errors. If you feel you want to add more contents or wish to be a part of Content Dev please contact us at **support@notespaedia.com**

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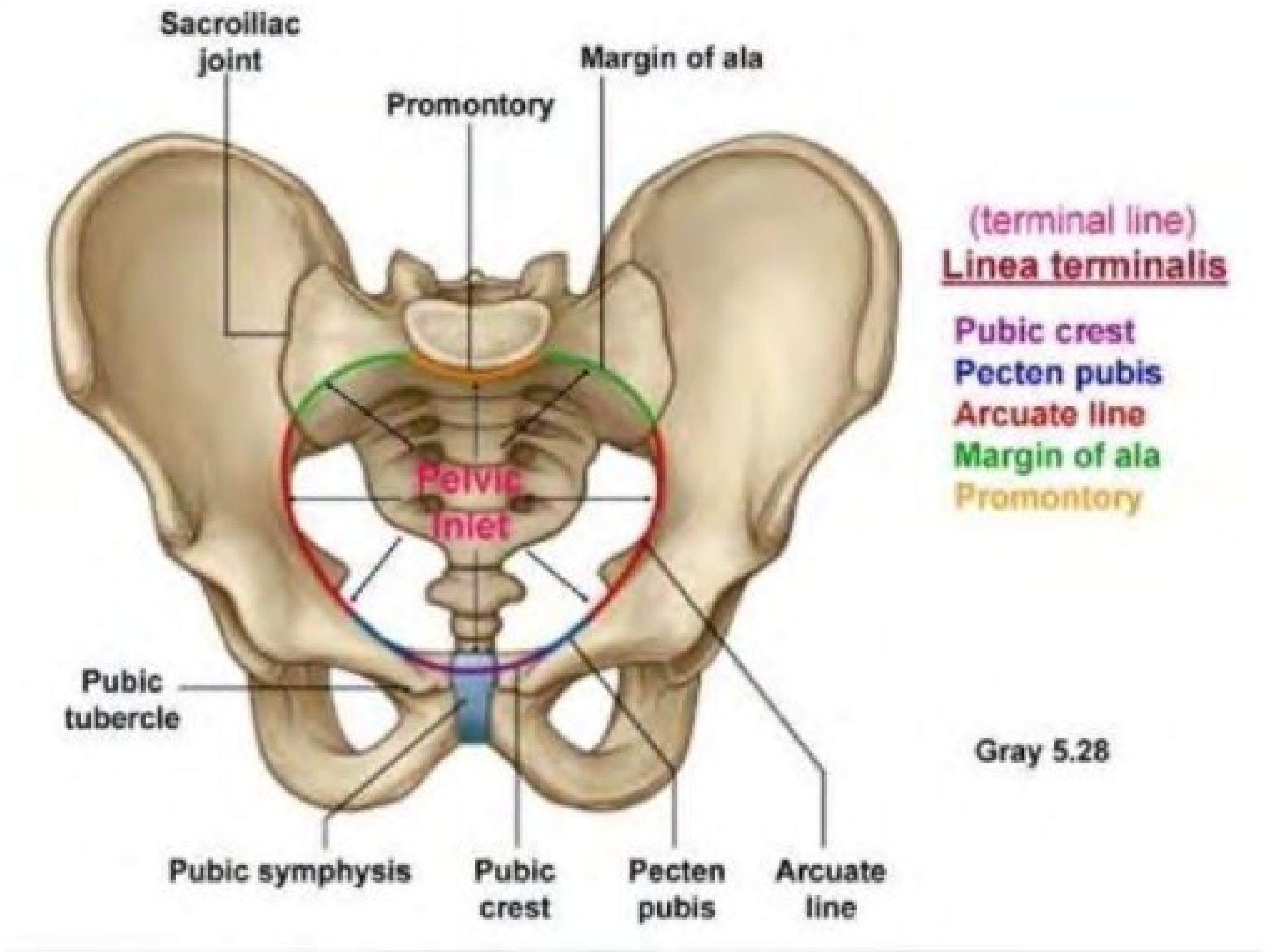
Pelvis and Fetal Skull

PELVIS

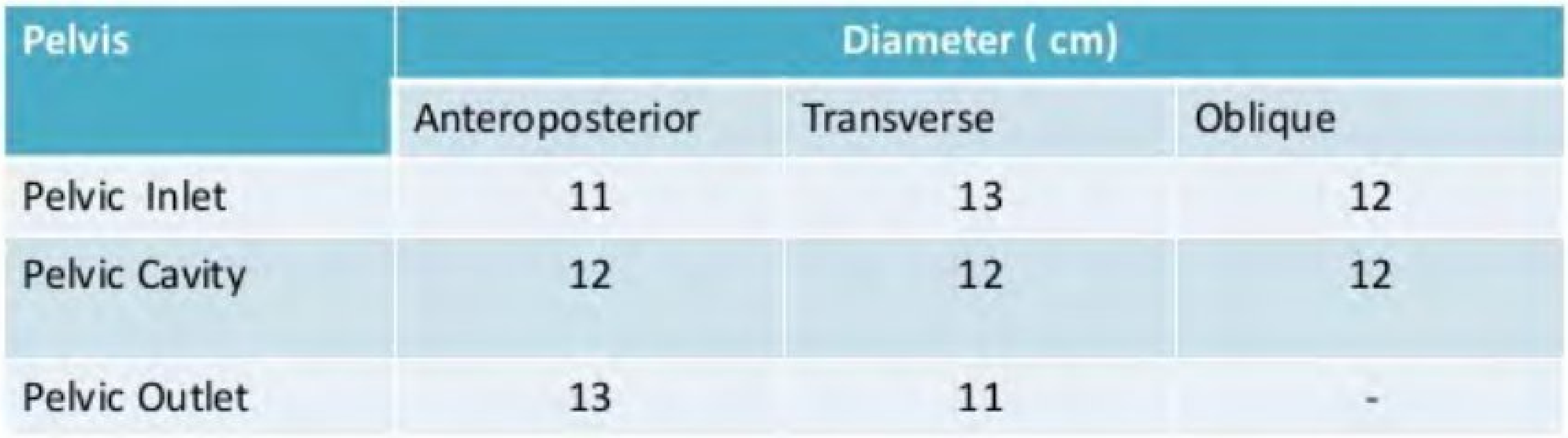
JOINTS IN THE PELVIS :-

- Symphisis Pubis → Fibrocartilaginous Joint
- Sacroilliac Joint → Synovial Joint
- Sacrococcygeal Joint → Synovial Hinge Joint

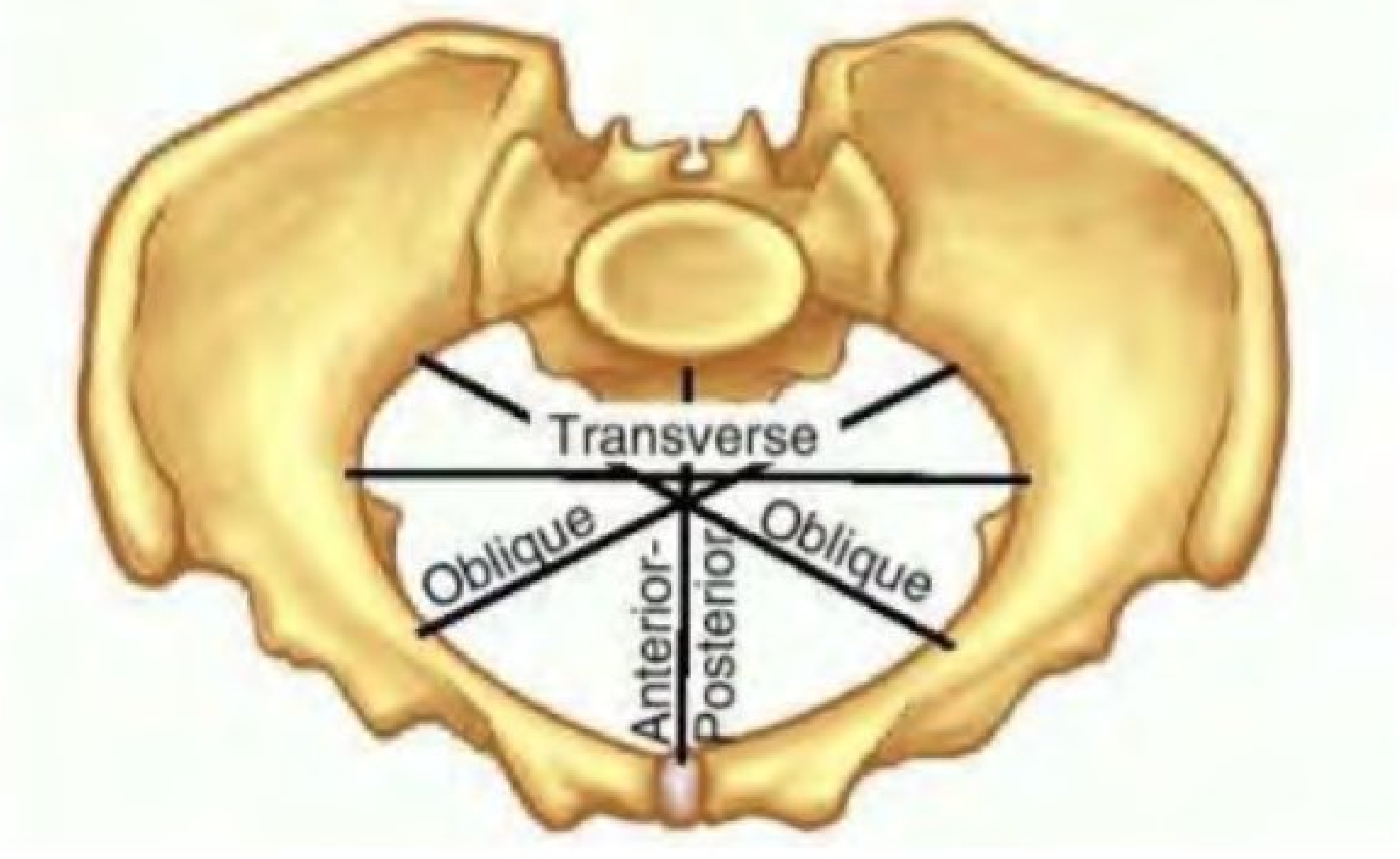


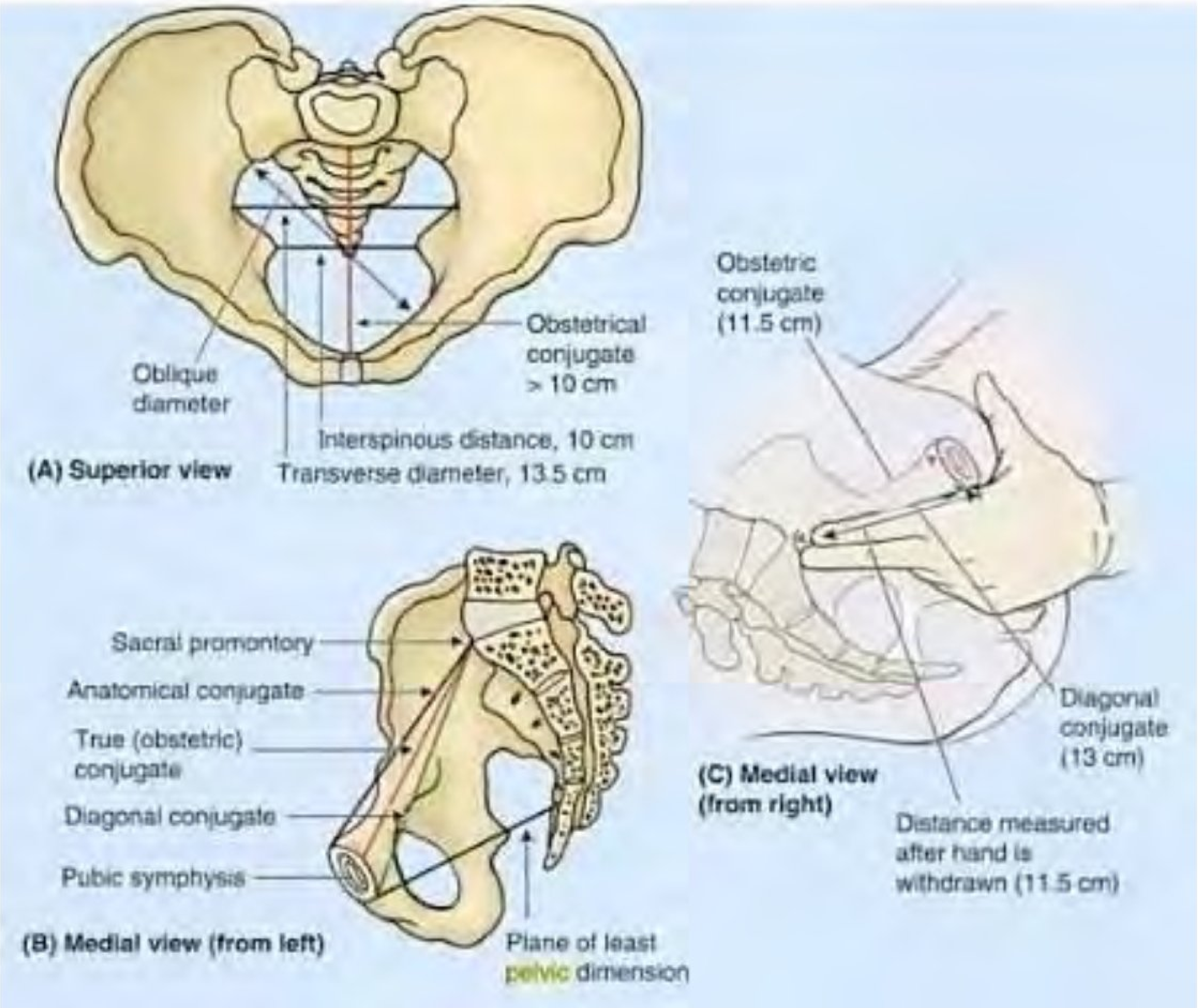
- False Pelvis → False palvis lies above the pelvic brim and has no obstetrics signifies
- True Pelvis → True pelvis lies below the palvic brim and plays an important role in childbirth and dalivery.

DIAMETERS OF PELVIS



Pelvis	Diameter (cm)		
	Anteroposterior	Transverse	Oblique
Pelvic Inlet	11	13	12
Pelvic Cavity	12	12	12
Pelvic Outlet	13	11	-




Oblique diameter
Obstetrical conjugate > 10 cm
Interspinous distance, 10 cm
(A) Superior view
Transverse diameter, 13.5 cm
Obstetric conjugate (11.5 cm)
Sacral promontory
Anatomical conjugate
True (obstetric) conjugate
Diagonal conjugate
Pubic symphysis
(B) Medial view (from left)
Plane of least pelvic dimension
Diagonal conjugate (13 cm)
(C) Medial view (from right)
Distance measured after hand is withdrawn (11.5 cm)

① TRUE CONJUGATE / ANATOMICAL CONJUGATE :
- 11cm
- measured from the midpoint of sacral promontory to the upper border of pubic symphysis.

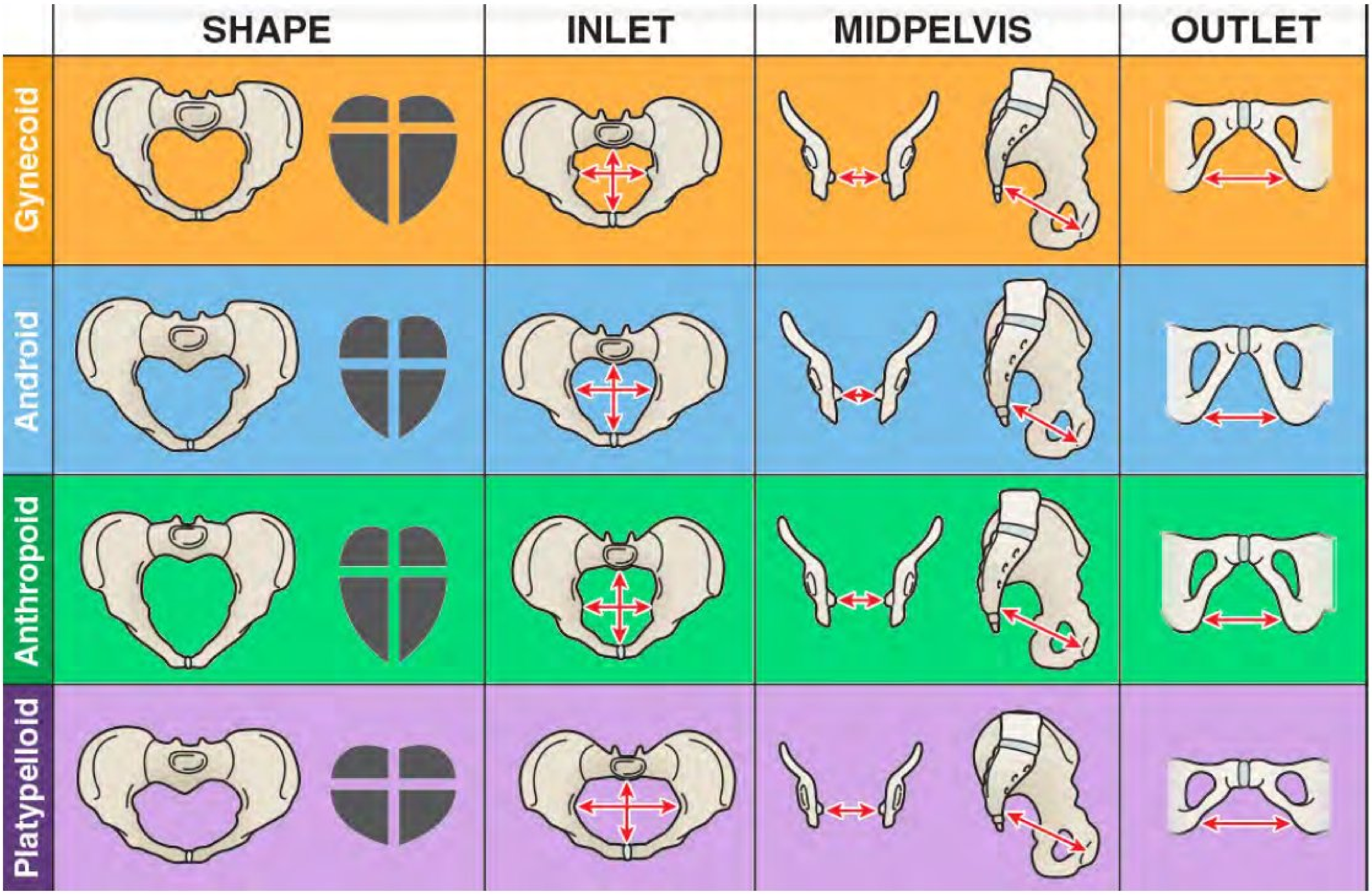
② OBSTETRIC CONJUGATE :
- 10 to 10·5cm
- Most important anteroposterior diameter of the pelvic inlet
- Fetus passes through this diameter
- Q: smallest A-P diameter
- Measured from symphysis pubis to the middle of sacral promontory

③ DIAGONAL CONJUGATE :
- 12·5cm
- Measured from the tip of sacral promontory to the lower border of pubic symphysis.

④ TRANSVERSE DIAMETER :
- 13 to 13·5cm
- Distance between the farthest two points on the iliopectineal line.
- Largest diameter of the pelvic inlet and lies 4cm anterior to the promontory and 7cm behind the symphysis

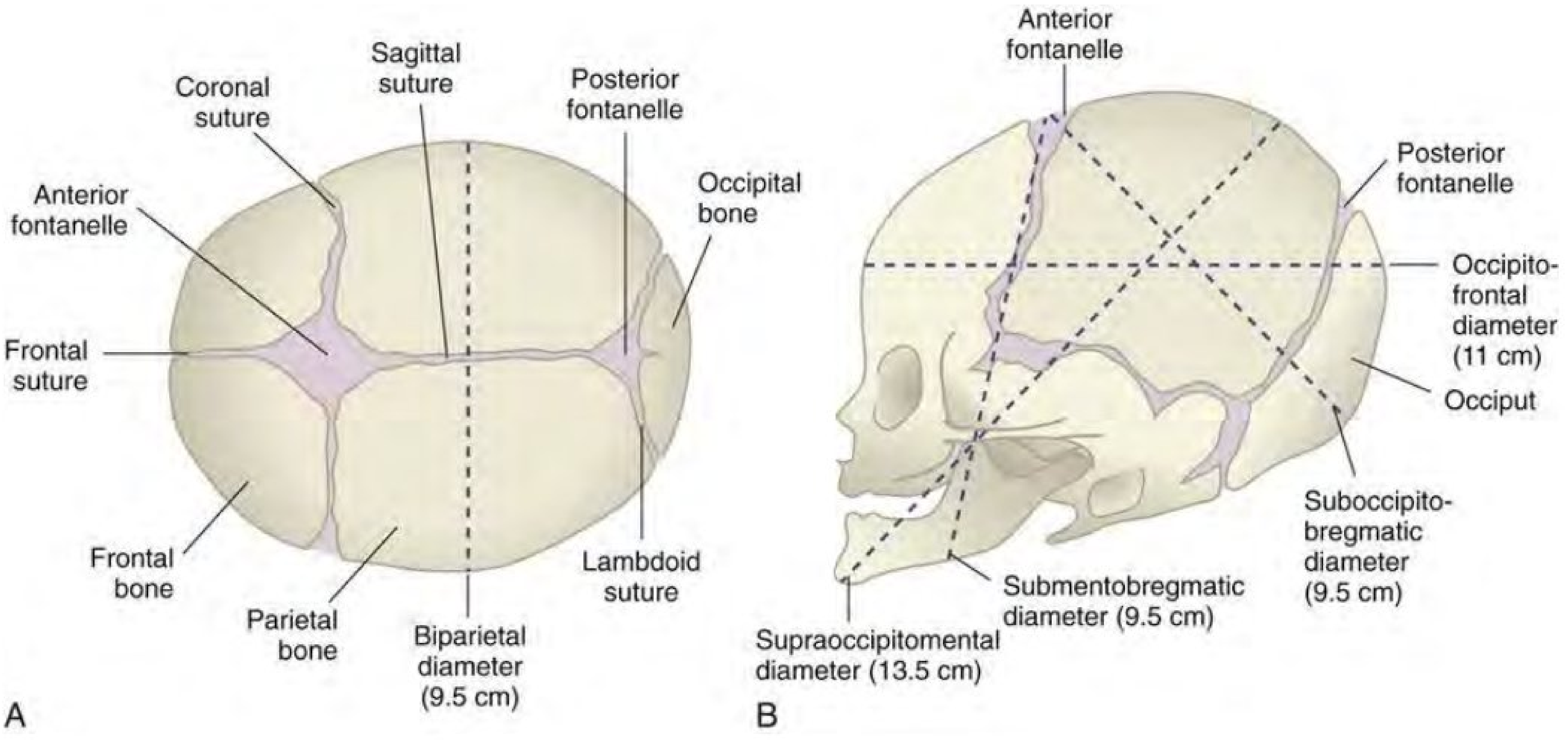
⑤ OBLIQUE DIAMETERS OF PELVIC INLET : • 12cm

PELVIS SHAPE



FETAL SKULL

SUTURES :

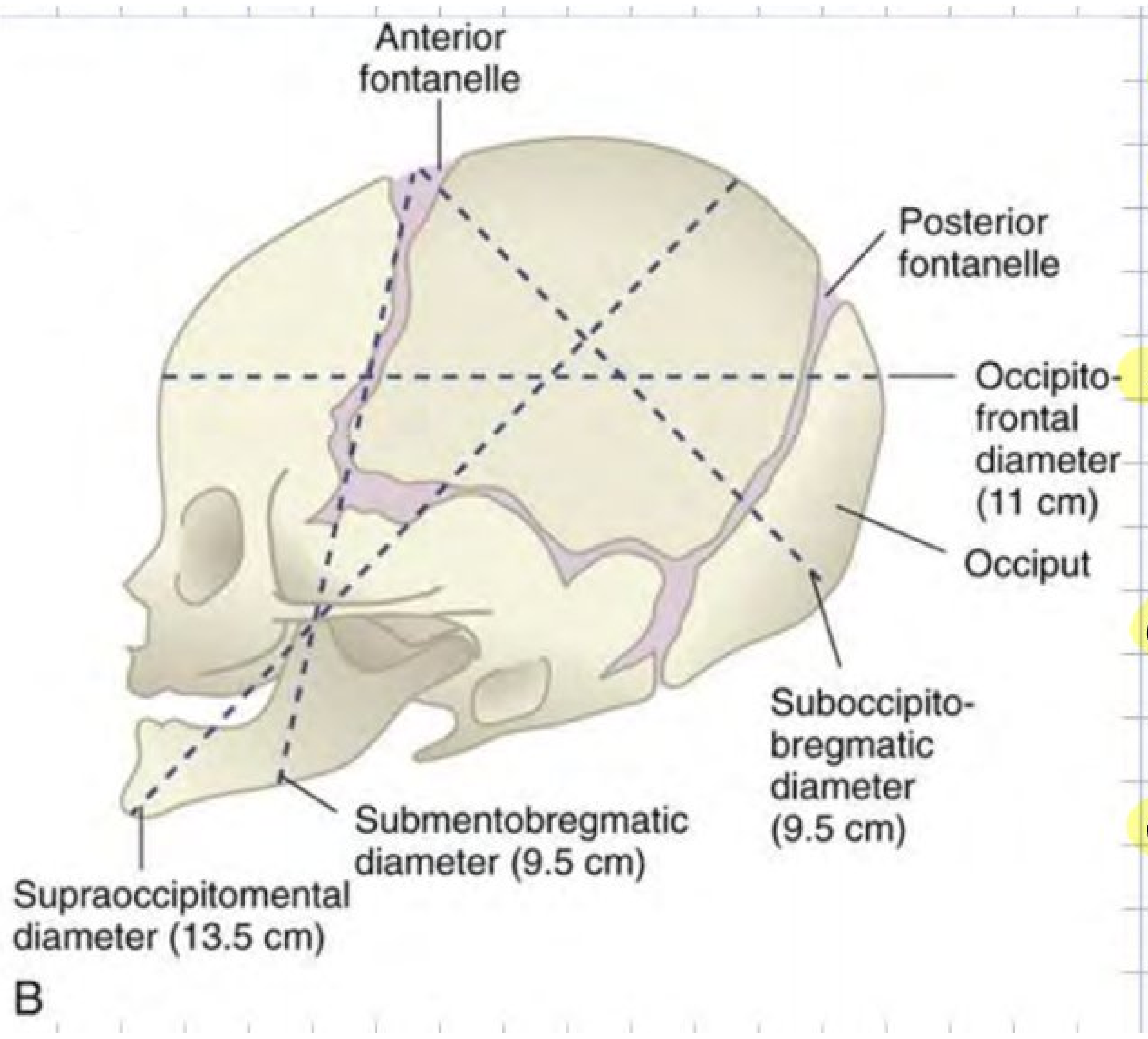


MOULDING: altaration of the shape of the fore-coming head while passing through the resistant birth passage during labor.

GRADING: There are 3 gradings:

- GRADE 1: Bones touch but not overlap
- GRADE 2: Overlap but easily seperated
- GRADE 3: Fixed overlapping

DIAMETERS OF FETAL SKULL:


Anterior fontanelle
Posterior fontanelle
Occipito-frontal diameter (11 cm)
Occiput
Suboccipito-bregmatic diameter (9.5 cm)
Submentobregmatic diameter (9.5 cm)
Supraoccipitomental diameter (13.5 cm)
B

TRANSVERSE DIAMETER:

① Biparietal Diameter → 9.5cm
· Extends between two parietal eminence.

② Supersubparietal diameter → 8.5cm
extends from a point placed below one parietal eminence to a point placed above the other parietal eminance of the opposite side

③ Bitemporal diameter → 8cm
· Distance between anteroinferior ends of the coronal sutures.

④ Bimastoid diameter → 7.5cm
· Distance between the tip of mastoid process

Q: If both ala of saccumb bone are absent, pelvis is called as:

a) Naegele Pelvis
[✓ b) Robert Pelvis]
c) Triradiate Pelvis
d) Rachitic Pelvis

sol. · One ala is absent - pelvis → NAEGRLE'S PELVIS
· Both ala of saccumb are absent - pelvis → ROBERT'S PELVIS
· Both are varieties of contracted pelvis and in both caesarean delivery is done.

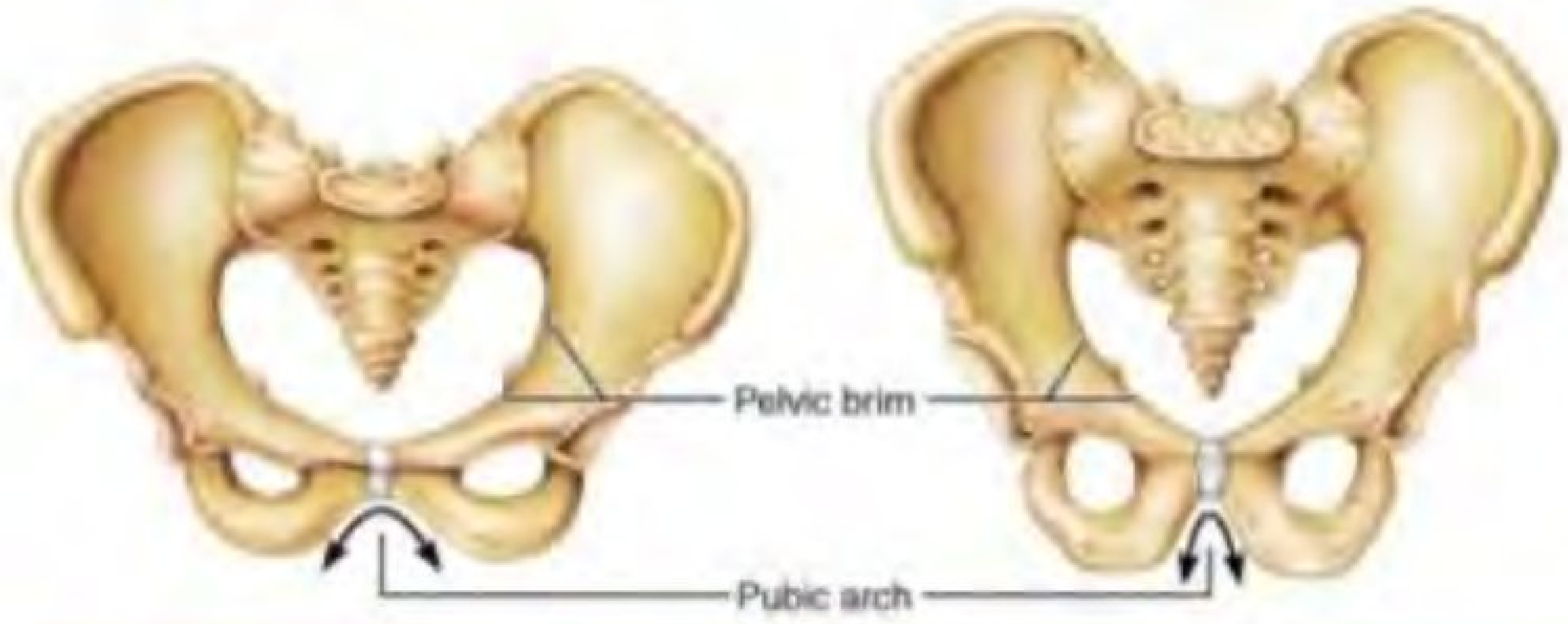
Q: CPD is best assessed by:

a) CT Scan
b) Ultrasound
c) Radio Pelvimetry
[✓ d) Pelvic assessment]

Q: Information obtained by lateral plate Xray pelvimetry are all except :
a) Sacral Curve
b) True conjugate
[✓ c) Bispinous diameter]
d) Inclination of the pelvis

Q: Which type of pelvis is associated with increased incidence of 'FACE TO PUBIS' Delivery?
a) GYNAECOID Pelvis
[✓ b) ANTHROPOID Pelvis]
c) ANDROID Pelvis
d) PLATYPELLOID Pelvis

DIFFERENTIATING B/W MALE & FEMALE PELVIS



FEMALE	MALE
Bones are lighter, thinner	Bones heavier, thicker
False pelvis is shallow	False pelvis is deep
Pelvic cavity is wide & shallow	Pelvic cavity is narrow & deep
Pelvic inlet round/oval	Pelvic inlet heart-shaped + smaller
Pelvic outlet comparatively large	Pelvic outlet comparatively small
Subpubic angle large	Subpubic angle more acute
Coccyx more flexible, straighter	Coccyx less flexible, more curved
Ischial tuberosities more everted	Ischial tuberosities longer, face more medially

Basics Of Reproduction

ANATOMY & FUNDAMENTALS OF REPRODUCTION

Female External Genitalia:

① MONS PUBIS → inverted Δle are above pubic symphisis, also called **MONS VENALIS**
Growth of this area → PUBARCHE ⇝ androgen dependant

SEQUENCE OF PUBERTY → Ⓖ Ⓣ Ⓐ Ⓜ

Ⓖ → ESTROGENIC { Begining of Growth Spur

Ⓣ → THELARCHE
↓
Breast Budding
[TANER STAGE II]
Estrogenic

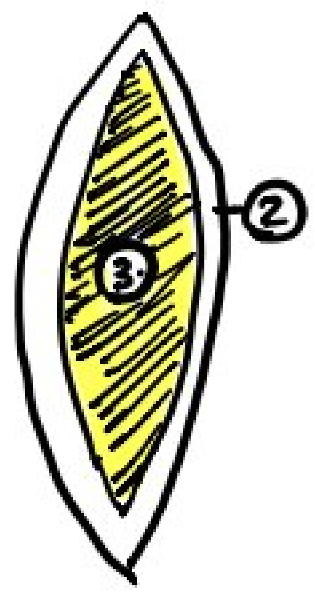
Ⓐ → ANDROGENIC
- Pubarche
- Adrenarche with peak growth velocity

Ⓜ → MENARCHE
- onset of menses } ESTROGENIC

② CLITORIS : origin : Genital Tubercle
Epithelium : Stratified Squamous Keratinised
Homologous ⇔ penis
Blood Supply = Deep and dorsal arteries of clitoris } from internal pudendal
& Lymphatic Drainage : DEEP INGUINAL LYMPHNODES {L·N of CLOQUET} → · Body · Glans · Crurae
SUPERFICIAL INGUINAL L·N → · Prepuce · Frenulum

# Sentinal L·N for vulval cancer → Inguinal L·N

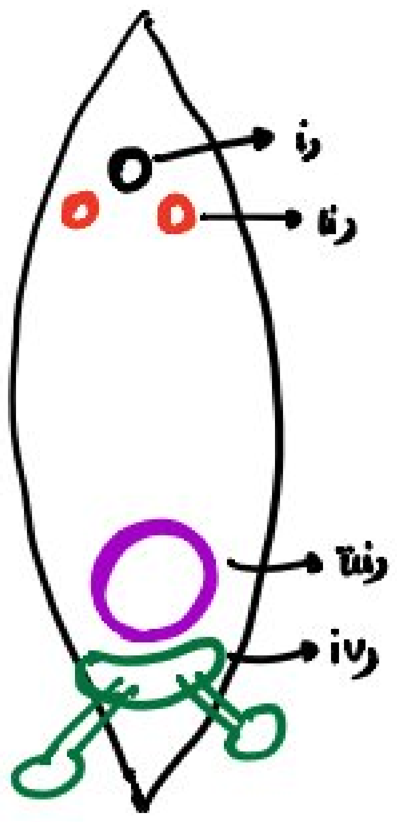
③ LABIA MINORA →
- originates : from genital folds / urethral folds
- homologous ↔ penile urethra & ventral part of penis
- L·N : Deep inguinal
- Epithelium : i) Stratified squamous non keratinised ⇒ medial to } HART'S LINE
  ii) Stratified squamous Keratinised ⇒ Lateral to



④ Vestibule : Opening in External genitalia
Origin : Urogenital Sinus

Opening in Vestibule :

i) Urethral Opening → origin : urogenital sinus
length : 4cm
Diameter : 6mm
Epithelium : upper $2/3^{rd}$ → Transitional epithelium
lower $1/3^{rd}$ → Stratified squamous non keratinised



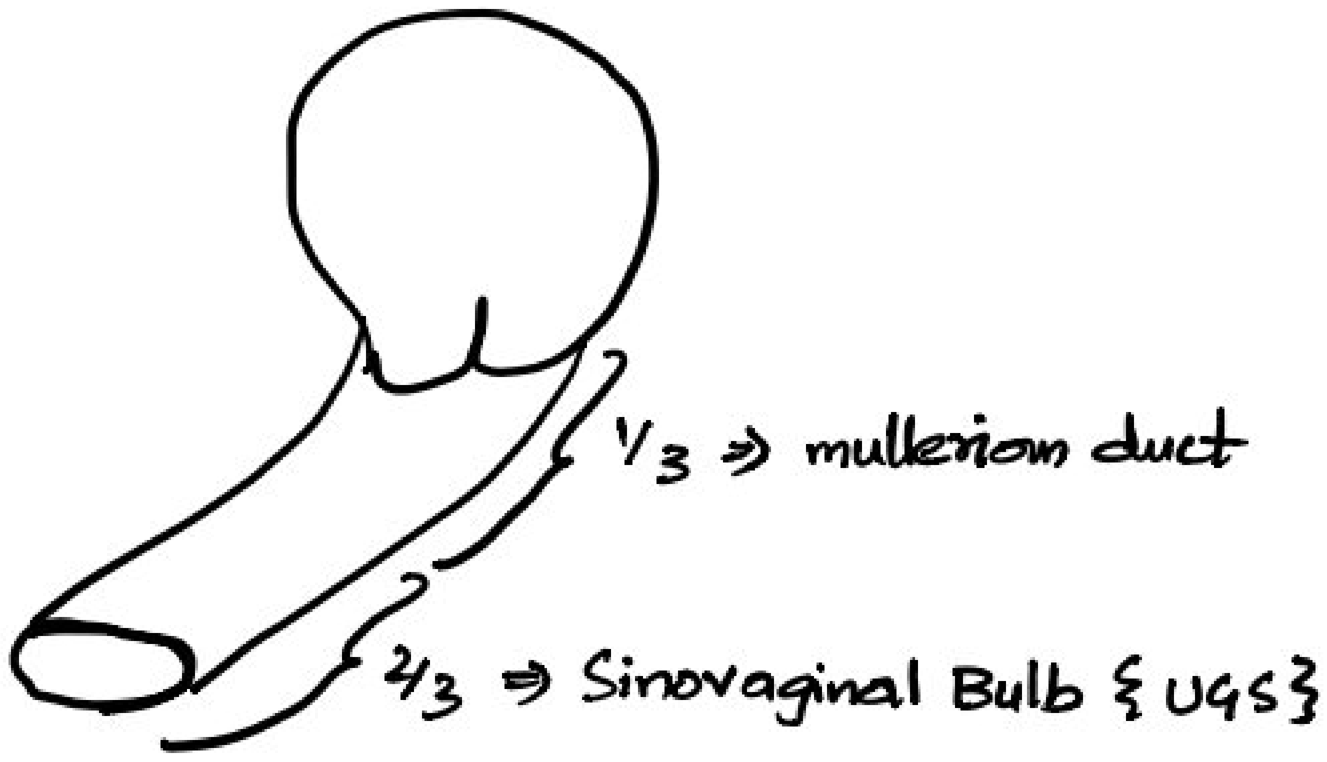
No. of opening = 6
Types of opening = 4

ii) Paraurethral gland / skene's gland → Location : 1'0 Clock } to vestibule
11'0 Clock

Homologous : Prostate in ♂
Origin : UGS
function : Lubrication

iii) Vaginal Opening → Origin : sinovaginal bulb {part of UGS}

Also forms
a) Lower $2/3^{rd}$ of vagina } upper $1/3^{rd}$ of vagina = Mullerian Duct
b) Hymen ← Remnant [TORN HYMEN] { Caruncule Myrtiformis
↳ Epithelium of complete vagina



iv) Fossa navicullar → carry Bartholins duct opening × 2

BARTHOLIN GLAND :
- compound racemose gland, 1cm in size homologous to COWPER'S GLAND
- also called as greater vestibular glands
- location : 5'0 Clock } in vestibule
  7'0 Clock
- situated in SUPERFICIAL PERINIAL POUCH
- pH → alkaline
- function → Lubrication (@ sexual excitement)

BARTHOLIN CYST : Posterior Lateral | SKENES CYST ⇒ Anteriol Lateral

Rx : Marsupialization (open + evert) { only for symptomatic patients
Asymptomatic cyst → No Rx. }

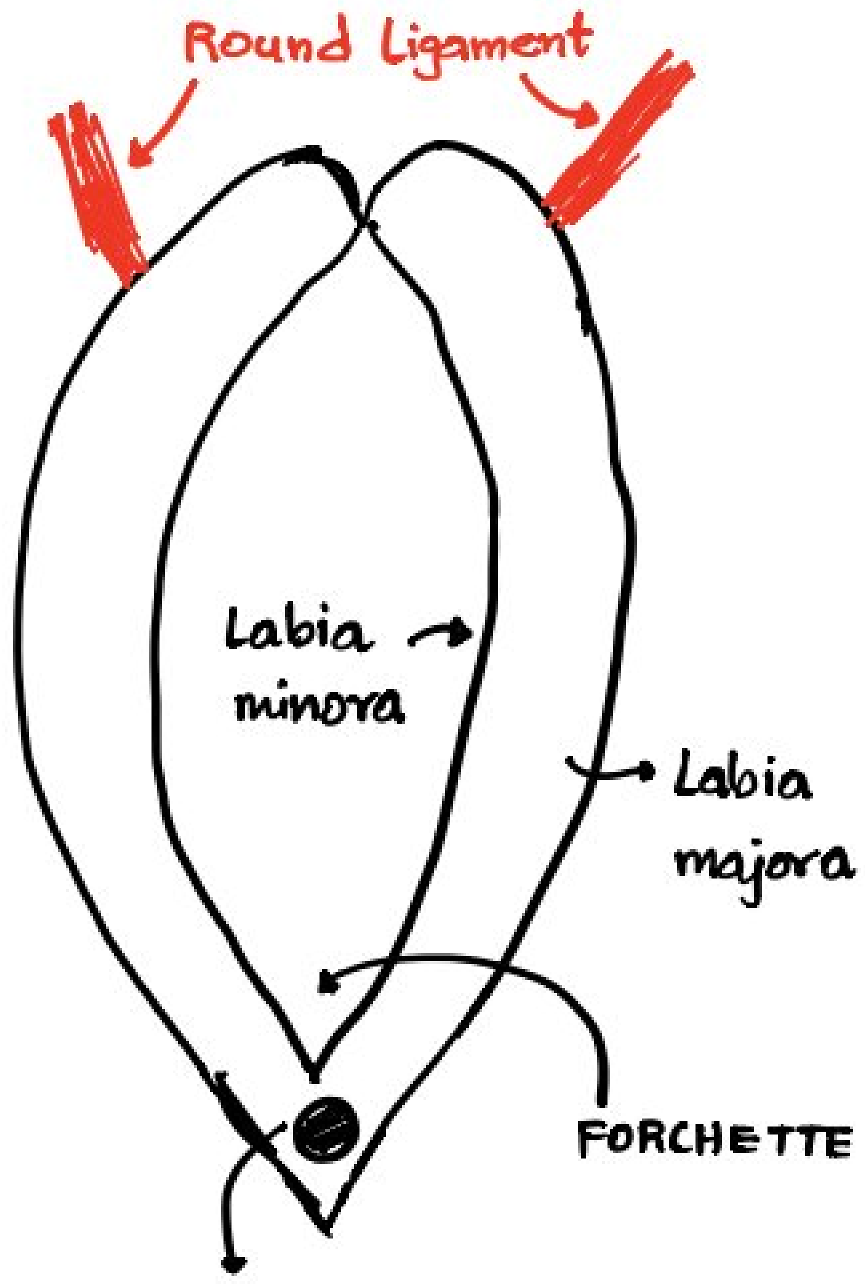
Barthdin Abscess : i, Pus formation

ii, I & D { symptomatic
or asymptomatic

④ **Labia Majora** ⇒ • origin ⇒ Genital swelling / gonodal swelling (Labio scrotal swelling)
- Homologues → Scrotum in ♂
- Will have ⇒

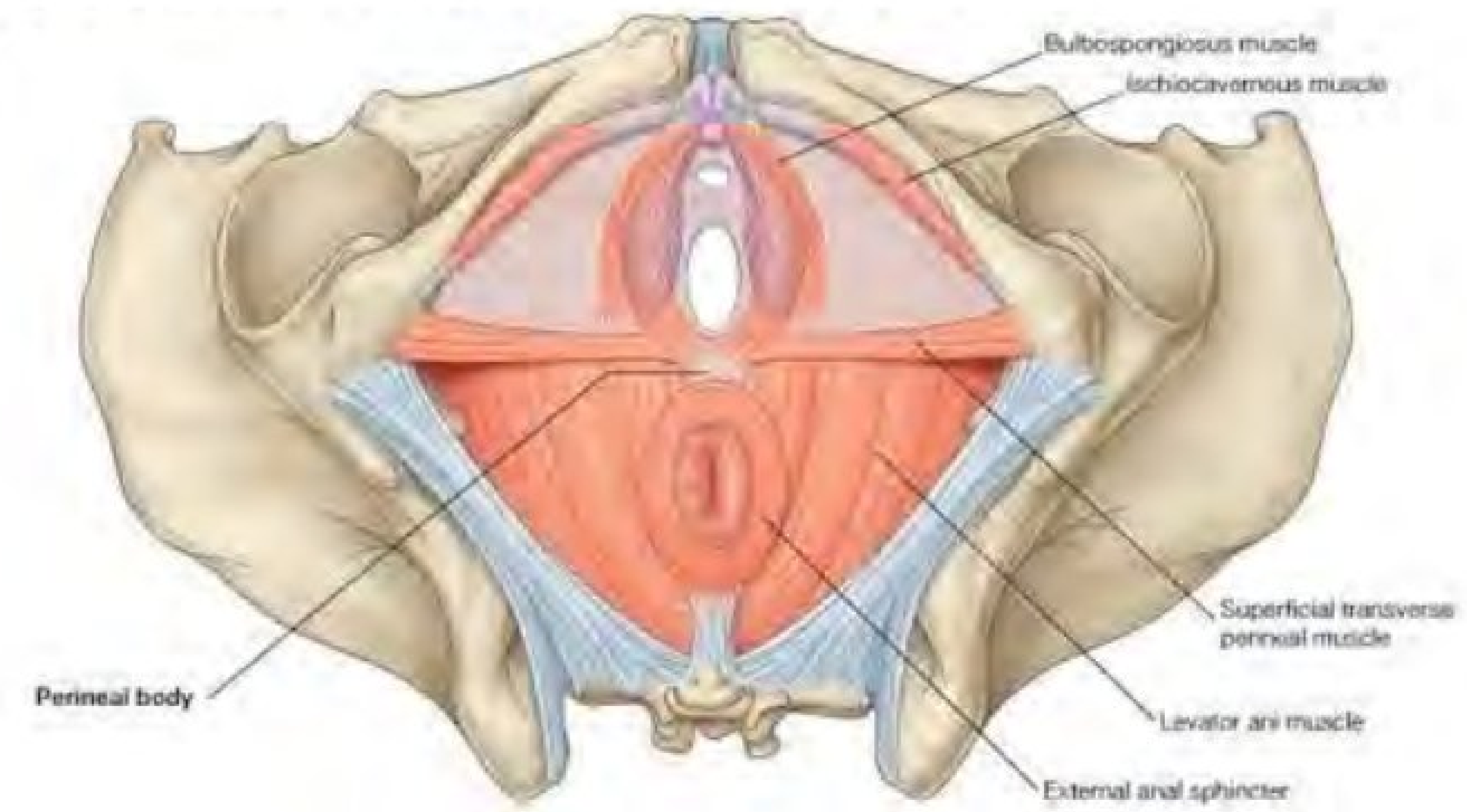
	Labia Minor
Apocrine glands →	×
Ecrine glands →	×
Sweat glands →	×
Sebaceous Glands →	✓
Hair follicle →	×

- LN → Superficial Inguinal Lymph node



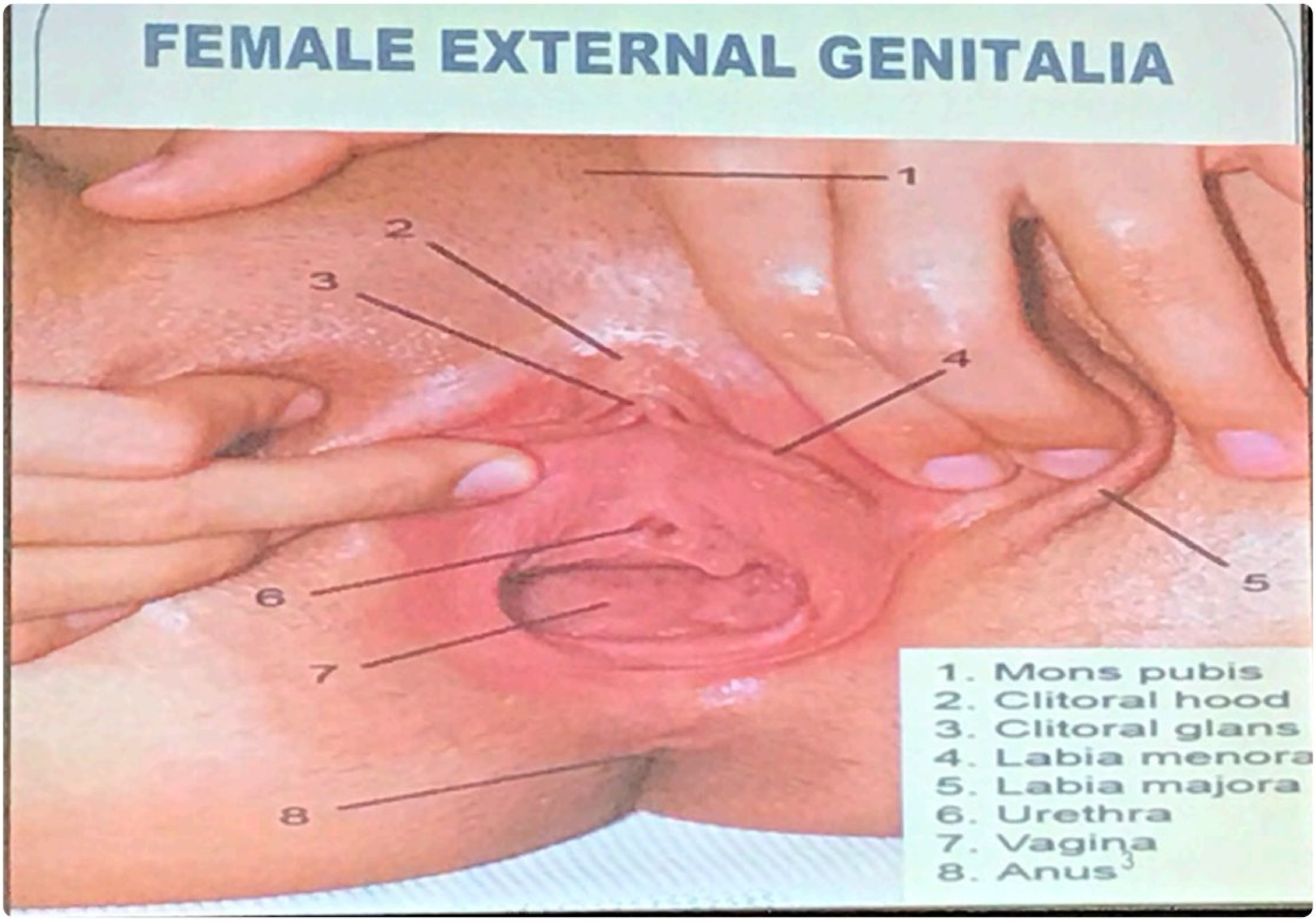
Posterior Commisure
above Perineal Body
→ Obstetric Perinium
fx : Support to internal organs

9 pelvic floor muscle meeting → i, Superficial Tranverse Perineii (2)
ii, Deep Transverse Perineii (2)
iii, Levator Ani (2)
iv, Bulbospongiosis (2)
v, External Anal sphincture



KIEGELS EXERCISE : prevents prolapse { not a treatment }

ROUND LIGAMENT → Homologue = Gubernaculum
↳ The ligament that pulls
down undescent testes to scrotum



FEMALE EXTERNAL GENITALIA

1. Mons pubis
2. Clitoral hood
3. Clitoral glans
4. Labia menora
5. Labia majora
6. Urethra
7. Vagina
8. Anus

SEX DETERMINATION

- It is decided by SRY gene → aka "SEX RELATED REGION Present on Y CHROMOSOME or TESTIS DETERMINING FACTOR"

  It is present on DISTAL END OF SHORT ARM OF Y CHROMOSOME

- If SRY gene PRESENT / Y CHROMOSOME PRESENT → SEX OF THE BABY - MALE

TURNER'S SYNDROME : 45 XO → Since Y chromosome lacking they are considered as Female.

KLINEFELTER'S SYNDROME → 47 XXY - Since Y chromosome present they are considered as Male

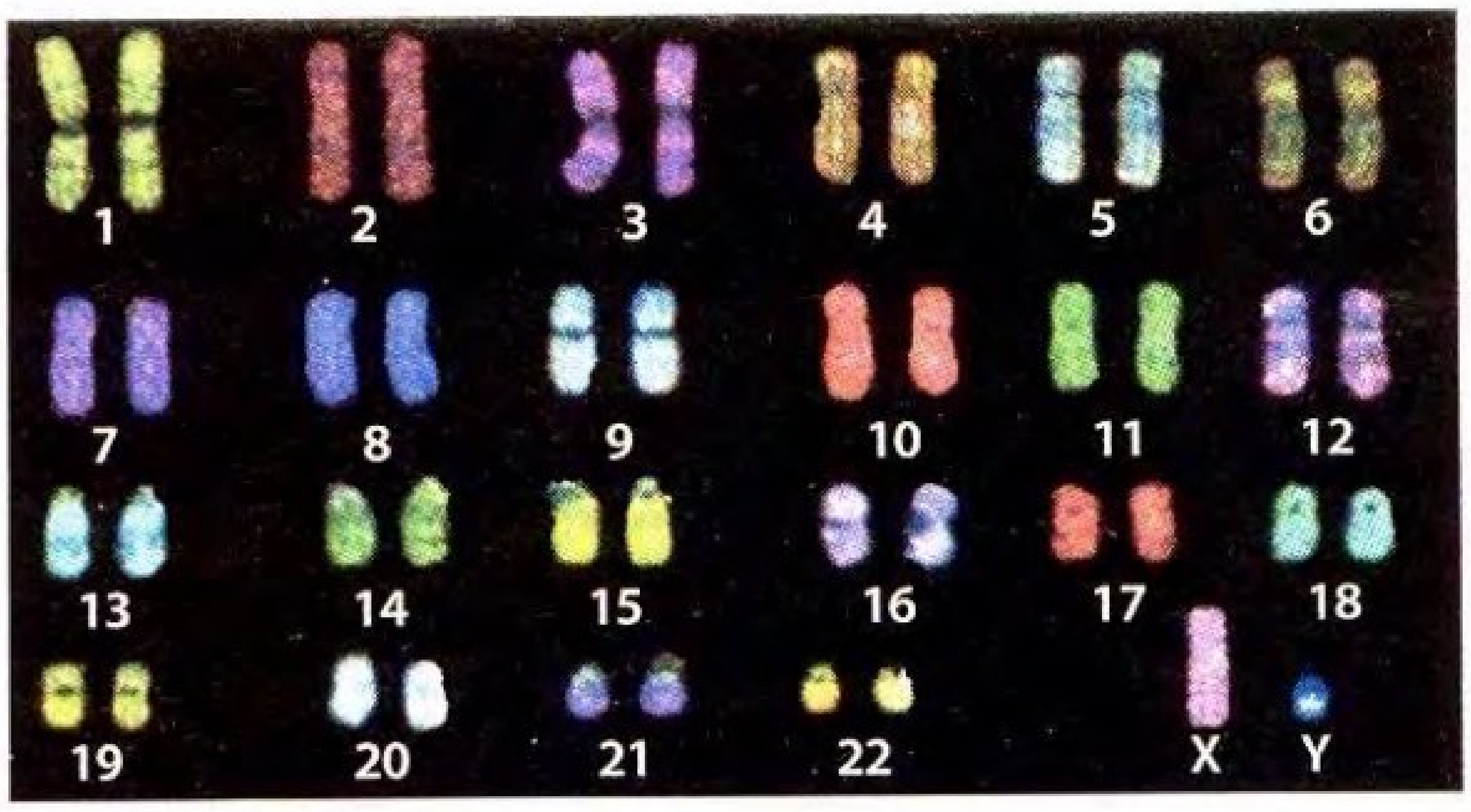
* Inspite of 2 Chromosome, still we consider as Male because of presence of Y Chromosome.

- Apart from main SRY gene, following genes helps in differentiation of testis and ovary they are ;

  1. Gene which favours Testis development → SOX - 9^{Q}

2. Gene which favours Ovary development → RSPO - 1
WNT - 4

BEST METHOD of Sex determination : KARYOTYPING

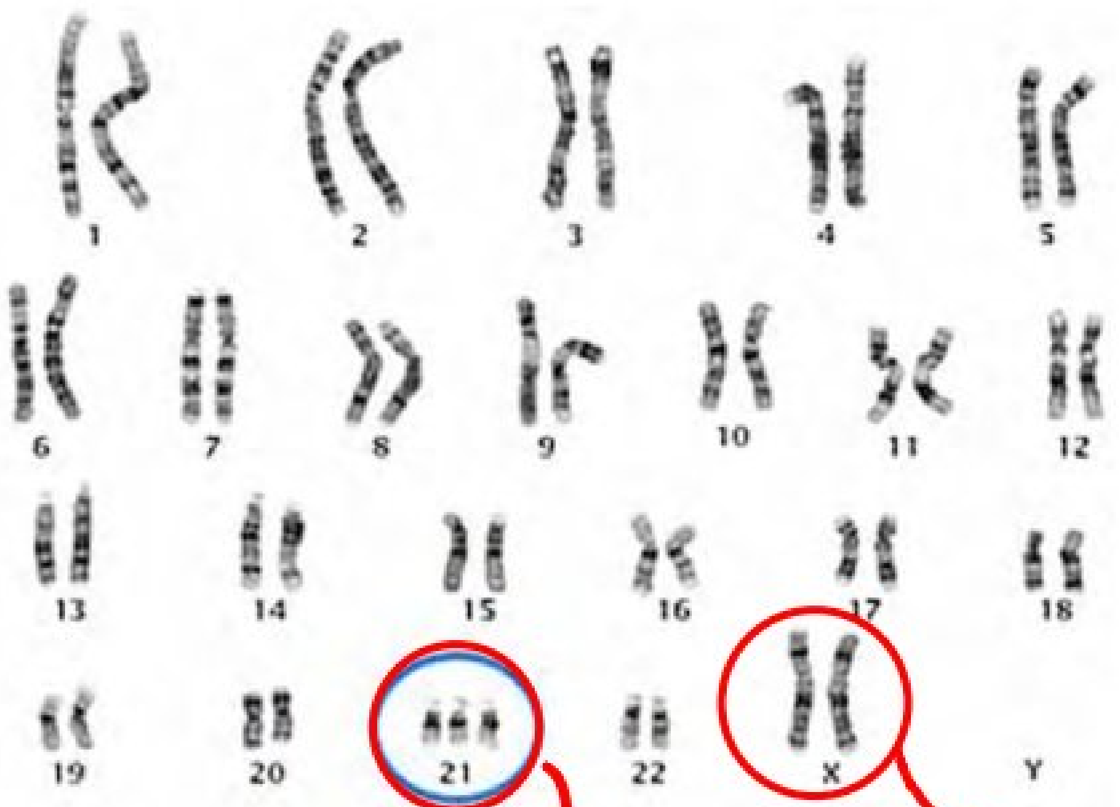


Karyotype of Normal Male

- It is best investigation for sex determination
- It is best investigation for detecting any defect in a chromosome number
  ↓
  K/a **ANEUPLOIDY**

ANEUPLOIDY

- **TRIPLOIDY** ↓ Chr. No. = 69
- **TRISOMY** ↓ Chr. No. = 47
- **MONOSOMY** ↓ One less chromosome Chr. No = 45



Karyotype Down Syndrome

Female

(MC) used for sex determination → Looking at External genitalia

AMBIGOUS GENITALIA - when sex of the baby cannot be determined by looking at External genitalia

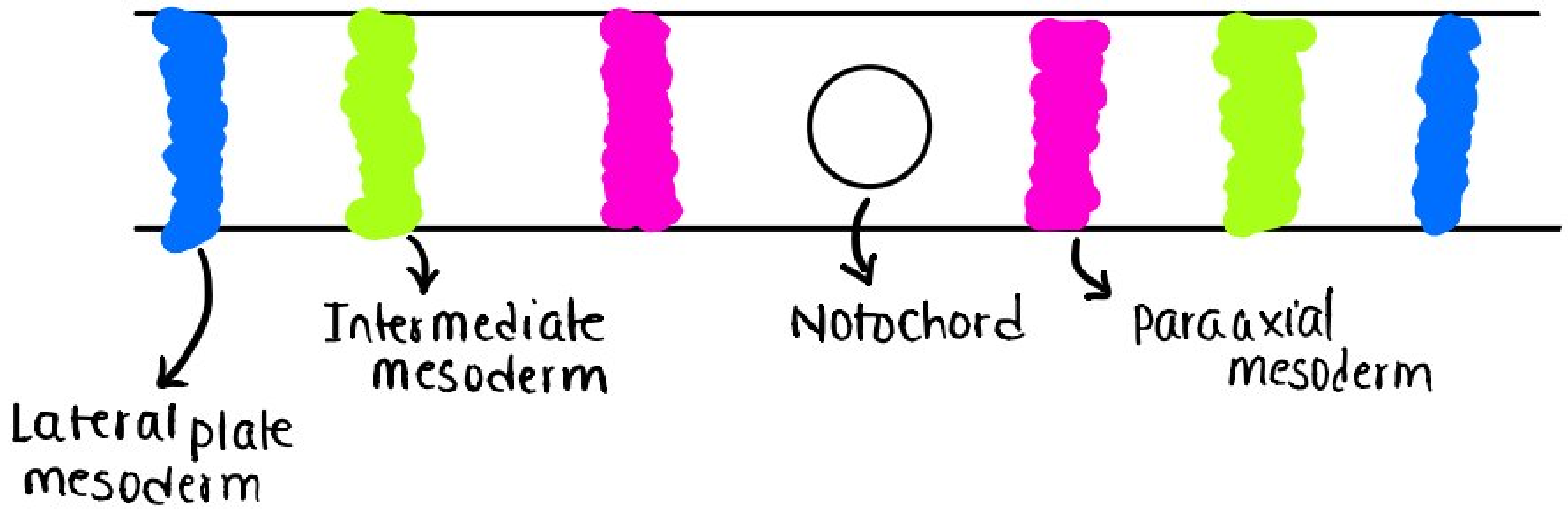
Other methods : of SEX DETERMINATION – is by counting number of BARR BODIES

[No. of Barr Bodies = Total no. of Chromosome - 1]

CONDITION	No. of BARR BODIES
1. Normal Male	0
2. Normal Female	1
3. Turner syndrome (45 XO)	0
4. Klinefelter Syndrome (47 XXY)	1

EMBRYOLOGY

- Gonads → Genital Ridge
- Ducts → Genital Ridge
- Germ cells → Epiblast
- External genitalia → Dorso somatic part of Lateral plate mesoderm



NOTE : → urogenital system → Most of it is mesodermal origin

Intermediate mesoderm → forms GENITAL RIDGE (5wks of Gestation)

- * Gonads
- * Ducts
- * Renal system

Lateral Plate Mesoderm → DORSOSOMATIC parts form → External genitalia

except → Those parts which develops from urogenital sinus → will be Endodermal origin

upper part of vagina → Mesodermal origin

Lower part of vagina → Endoderm origin

GONADS - till 6wks are BIPOTENTIAL → They can either differentiate into Male gonads or Female gonads

- It develops from Genital Ridge (In Mullerian Agenesis → ovaries will be Ⓝ) Since ovaries develops from Genital ridge
- differentiation of gonads depends upon : SRY Gene
  - Present → 'Y' Chromosome ⊕
  - Absent → 'Y' Chromosome absent

- Medulla of genital ridges forms testis
- Testis formed by 7wks

- Cortex of genital ridge forms ovary
- ovary formed by 8wks

The first feature which differentiates b/w Testis and ovaries is Formation of Testicular cords.

CONCEPT :

1. Ovaries are formed d/t absence of 'Y' chromosome

   In TURNER's Syndrome → Gonads → OVARIES

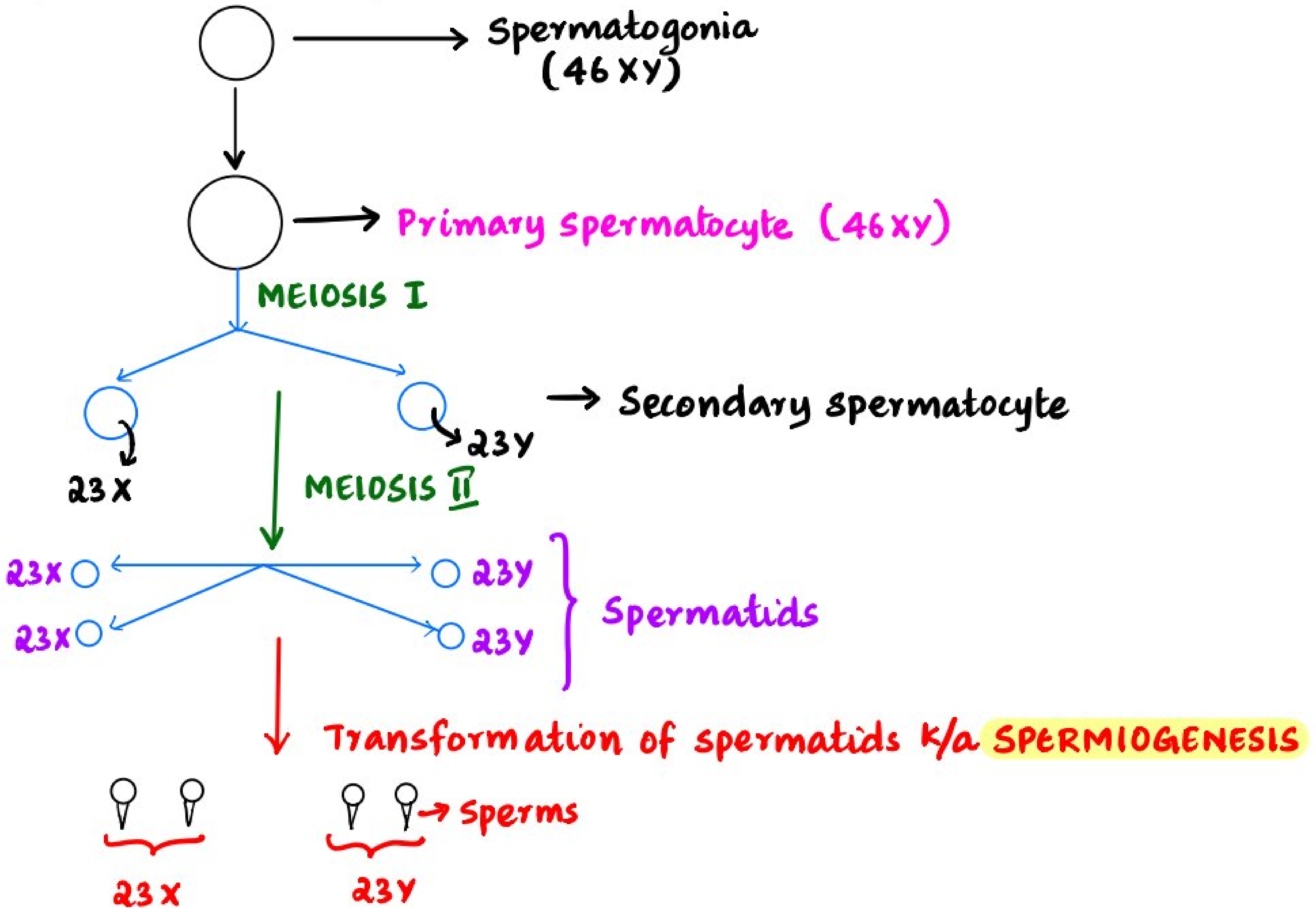
2. BUT → for proper development of ovaries both 'X' chromosomes are needed

   In TURNER's Synd (45 XO) = Ovaries are not properly developed k/a 'STREAK GONADS'

GERM CELLS

- Originates from EPIBLAST
- They are also BIPOTENTIAL
  - 'Y' Chromosome present → forms SPERMATOGONIA 46 XY or 44 + XY
  - 'Y' Chr. Absent → forms Oogonia 46 XX or 44 + XX

SPERMATOGENESIS



IMPORTANT POINTS ON SPERMATOGENESIS

1. Begins at puberty

2. * From 1 primary spermatocyte → 4 sperms are formed
   * From 1 Spermatogonia → 16 Primary Spermatocytes are formed
   * From 1 Spermatogonia → 16 x 4 = 64 sperms are formed

3. Time taken for Spermatogenesis → 72-74 days

   (Time in ductal system before ejaculating → 14-16 days)

4. Avg. Number of sperms produced per day → 100 millions/day

SPERMIOGENESIS

* Transformation of spermatids into sperms
* NO MITOSIS / NO MEIOSIS
* Time Taken : 12-14 days

Parts in Spermatids	In Sperms
Nuclear materials	Heads
Golgi Body	Acrosomal caps
Mitochondria	Middle piece
Microtuble/Centriole	Tail/Axial filament

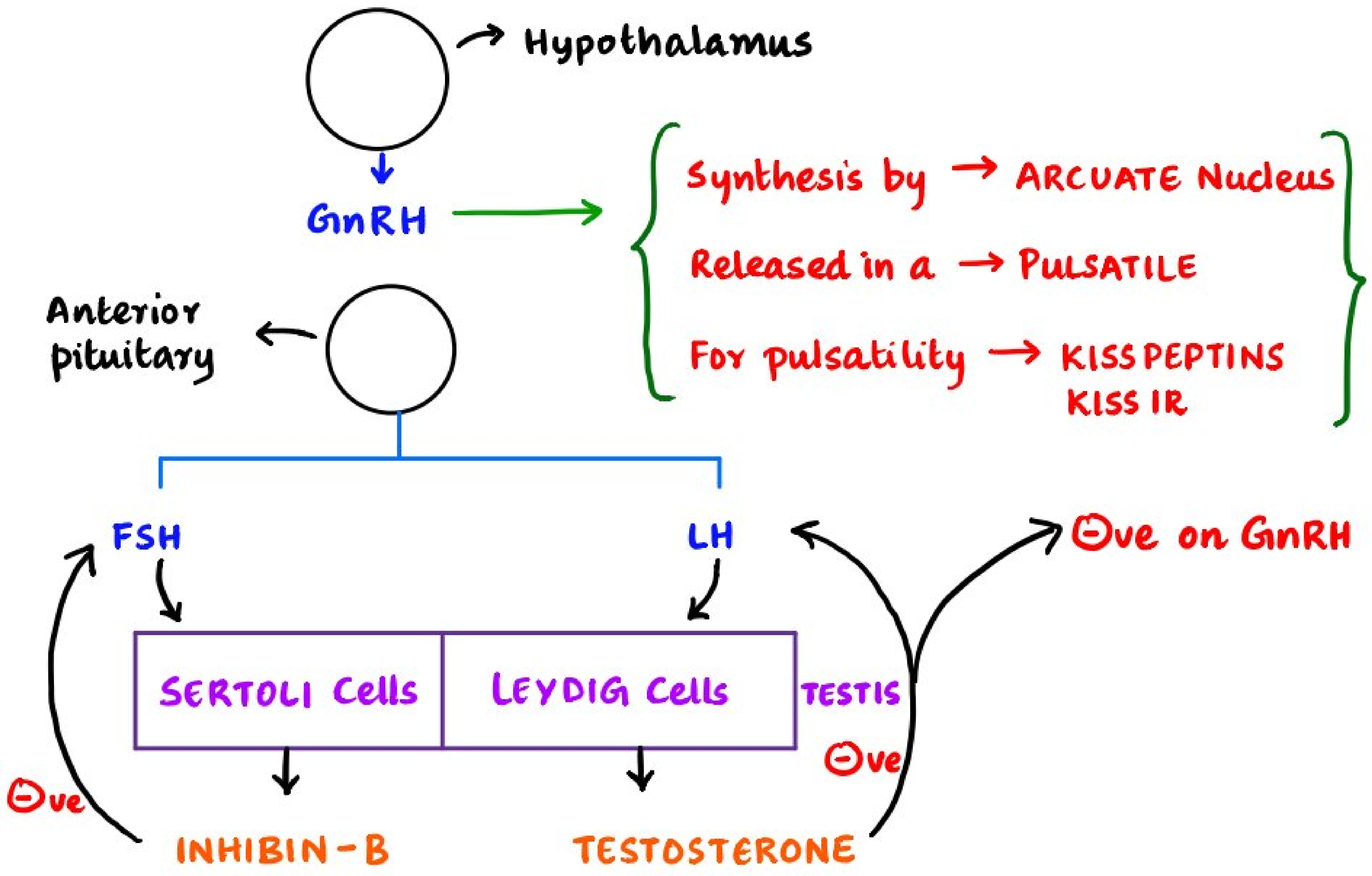
9 + 2 Arrangement

NOTE : sperms lack Endoplasmic Reticulum especially ROUGH ER

SPERMS

* Length → 55 microns
* Fertiliazable span → 48-72 hrs
* Sperms attain motility and maturity } Caudal end of Epididymis
* Genes for motility - CATSPER
* Ion for motility - Calcium
* Sperms remains motile in Female Genital tract } 12 hrs
* Time taken by sperms to reach ampulla of tube } 30 mins

HORMONAL SUPPORT OF SPERMATOGENESIS

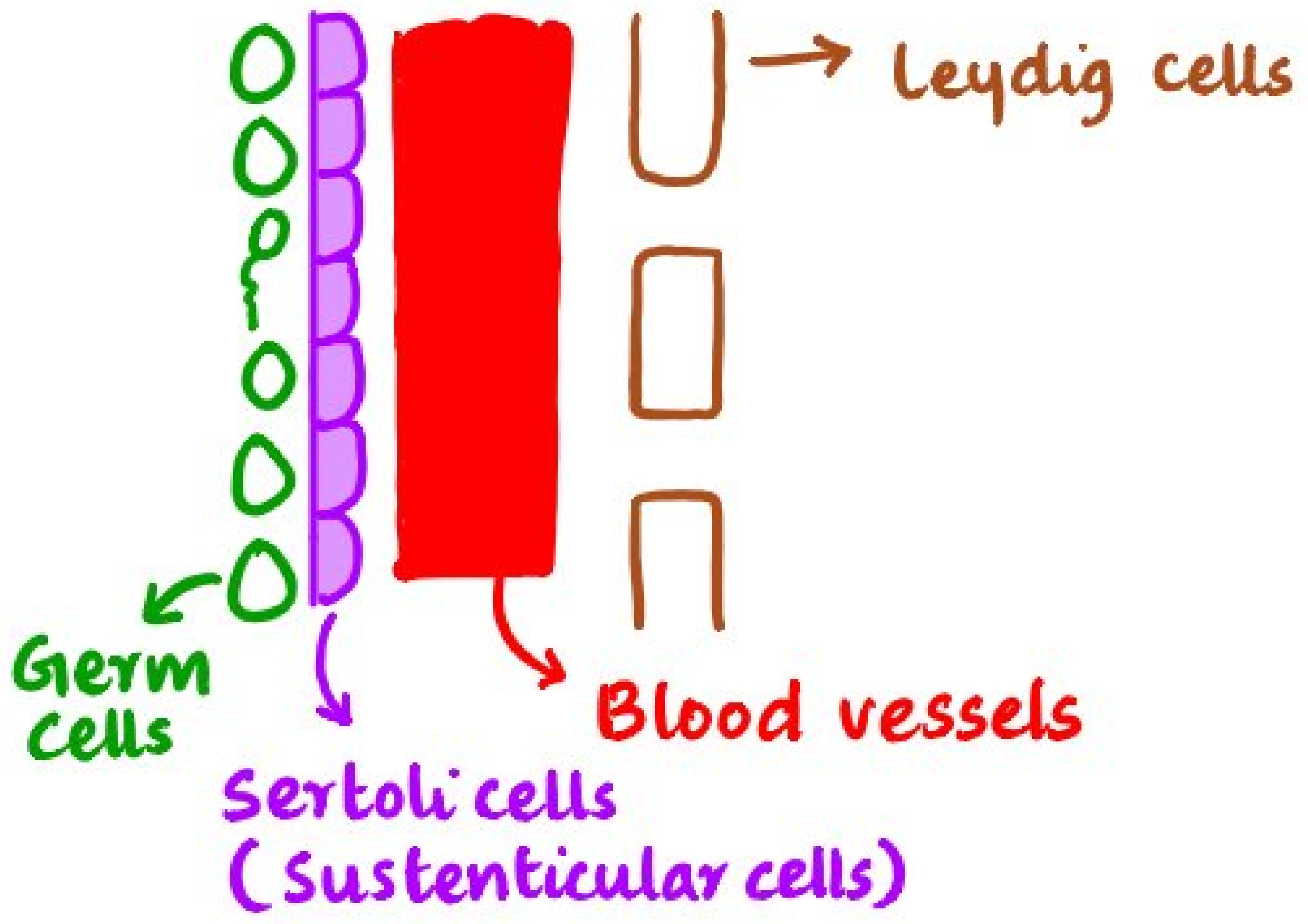


Q. Main Hormone needed for spermatogenesis → TESTOSTERONE

LH Receptors in Male → LEYDIG Cells

FSH Receptors in Female → SERTOLI Cells

Arrangement of Cells



Q. Blood testis Barrier is formed by
SERTOLI CELLS

Q. Cells lying outside blood testis
barrier is LEYDIG CELLS

OOGENESIS : Begins in IntraUterine Life

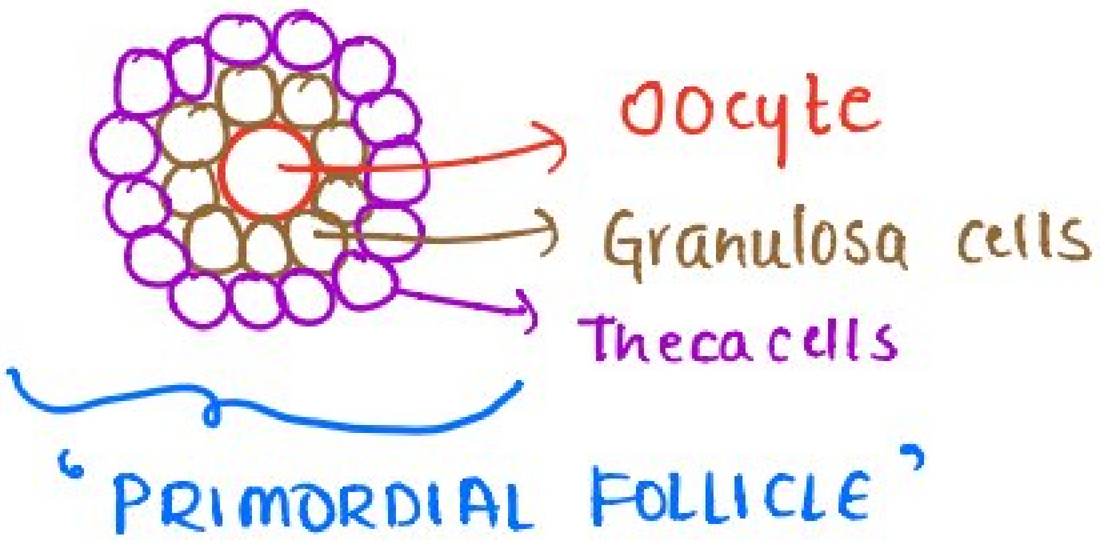
→ OOGONIA 46XX or 44+XX

↓ MITOSIS

→ PRIMARY OOCYTE 46 XX

↓ MEIOSIS 1 but it gets arrested in DIPLOTENE Stage of PROPHASE

- It remains arrested till puberty
- The arrested state is K/a DICTYATE STATE



'PRIMORDIAL FOLLICLE'

IN SPERMATOGENESIS
↓
DICTYATE STATE is ABSENT

- the primary oocyte gets surrounded by follicular cells which are

Granulosa cells, present in cortex of ovary and Theca cells, present in Medulla of ovary

"OVARY IS A RESERVOIR FOLLICLE"

At Puberty : HP ovarian axis gets activated → LH released from Anterior Pituitary

↓

LH Surge

↓

MEIOSIS I Resumes [Meiosis I is Hormone dependant (LH)]

↓

→ Primary Oocyte 46XX

OVULATION { Secondary Oocyte ← 23X

→ 1st polar body released 23X

↓ MEIOSIS II : But gets arrested in → METAPHASE Arrest

arrest is over at the time of fertilization

Female Pronucleus (23X)

2° Polar Body Released (23X)

OVULATION : → Release of 2° Oocyte from 1° Oocyte

LH Surge → MEIOSIS 1 Resumed (at 32-36hrs before Ovulation)

↓

OVULATION

- Time interval b/w LH Surge and Ovulation is
  - 32 - 36hrs (Best)
  - 24 - 36hrs (2nd Best)

* 1st Polar body is released at the time of OVULATION

Q. 1st Polar Body is released at

a. 24hrs before ovulation

b. 48hrs before Ovulation

c. at time of ovulation

d. 24 hrs after ovulation

* 2nd Polar Body is released at the time of Fertilization

IMPORTANT POINTS ON OOGENESIS

1. Begins at → Intrauterine life
2. Size of ova → 120 microns (ova = female pronucleus)
3. Size of Resting follicle → 0.02 mm
4. Size of follicle just before ovulation → 18-20mm
5. Fertilizable span of ova → 12-24 hrs

TIME TABLE OF EVENTS

1. Germ cells reach yolk sac → 3wks
2. Germ cells reach gonads → 6wks
3. Oogonia formed → 9wks
4. Primary Oocyte formed → 12-15wks
5. Follicle formation begins by → 15-20wks
6. Follicle formation is completed by → 22-24 wks

NUMBER OF FOLLICLES

1. Max^{m} no. of follicles seen at 5th month IUL → 6-7 million follicles

2. At birth → 1-2 million follicles

3. At puberty → 4-5 lakhs → Only 500 follicles mature in entire life time

(1000 follicles undergoes atresia every month)

Q

* Initial Recruitment of follicles is HORMONE DEPENDENT

TESTOSTERONE IN MALES

* Testosterone productions begins at 7-8 wks of IUL

↳ Maximum at 15 wks of IUL $\xrightarrow{S/i}$ 2nd TM → Then decrease

↙ Remains at low level till puberty

* At 17 yrs of age, testosterone = To Adult Male

- Testosterone in Males = 5-7 mg/day
  - ↳ Free testosterone - 0.5 to 3%

- Remaining Bound = mainly to sex hormone binding Globulin and Albumin

* Testosterone converted into → ESTRADIOL

→ DHT (Dihydrotestosterone)

Testosterone $\xrightarrow{\text{Aromatase}}$ Estrogen → Amount = 50mcg/day

20% produced in testis and rest in adipose tissue

FATE OF ESTROGEN IN MALES

1. ↑ Bone Mineral density
2. Needed for closure of Epiphysis of long Bones
3. Needed for maintaining Body fat
4. For sexual dysfunction

- IF ESTROGEN ↑ in males leads to GYNAECOMASTIA

Testosterone $\xrightarrow{5\alpha \text{ Reductase}}$ DHT : is the most potent androgens

* Testosterone : DHT Ratio → 10 to 15 : 1

Questions :

Q. Max^m Oogonia can be seen in ovaries at

a. 5th month of IUL b. at birth c. at 7th month of IUL d. at puberty

Q. 1st Meiotic division of oogenesis get arrested at

a. Pachytene stage of Prophase c. Leptotene stage of prophase

b. Diplotene stage of Prophase d. Metaphase stage of prophase

Q. In which of the following transition Meiosis occurs

a. Primary to secondary spermatocyte → MEIOSIS 1

b. Secondary spermatocyte to globus pallidus → MEIOSIS 2

c. Germ cells to spermatogonium

d. Spermatogonium to primary spermatocyte – MITOSIS

Q. Which is the first stimulus for Leydig cells to produce testosterone?

a. LH b. FSH c. HCG d. GnRH

Q. Time taken for Spermatogenis is

a. 50-60 days b. 60-70 days c. 70-80 days d. 80-90 days

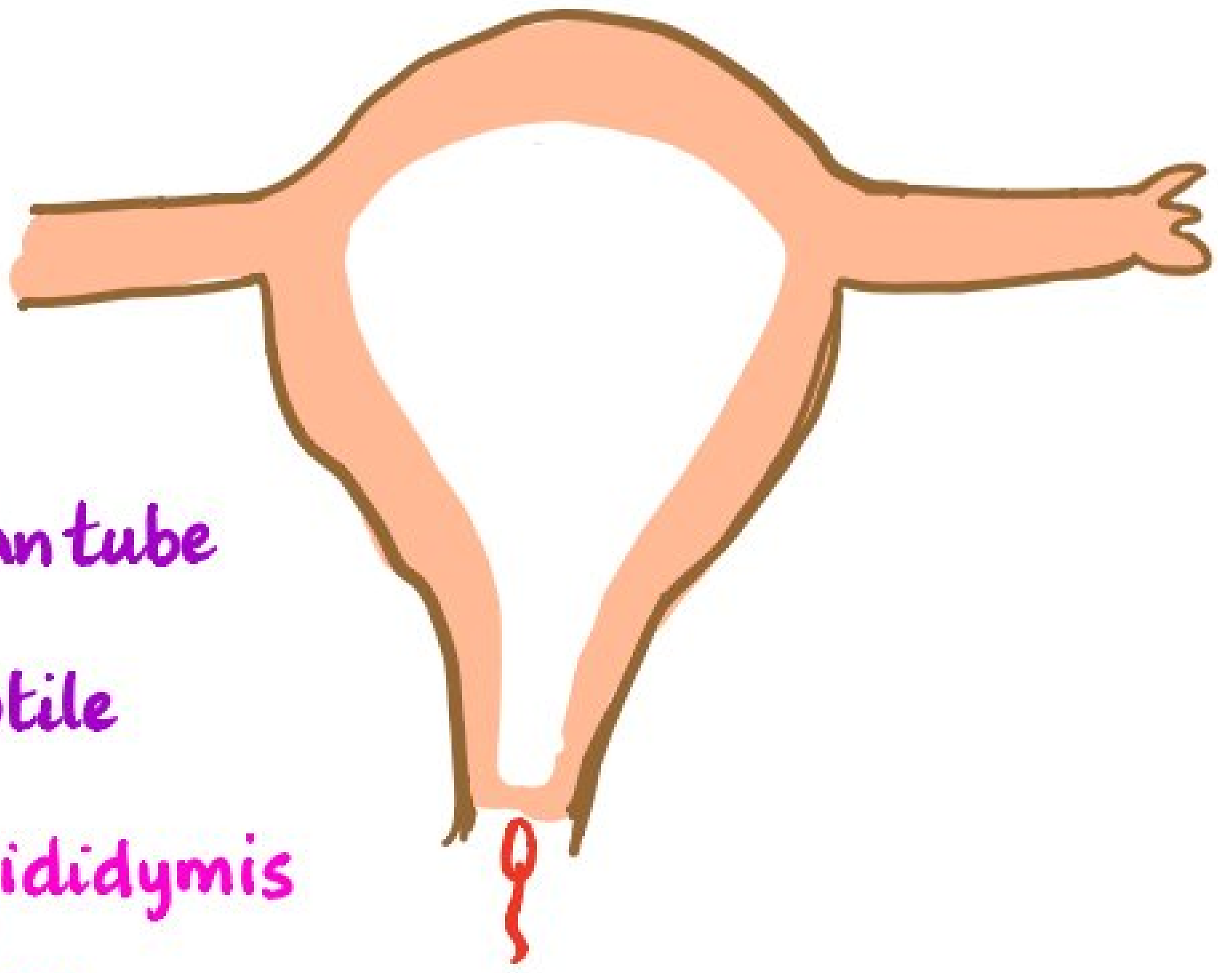
Q. The Target gene for SRY which favours testis differentiation:

a. SOX 9 b. RSPO 1 c. DAX 1 gene d. WNT 4

$\underbrace{\text{c. DAX 1 gene} \quad \text{d. WNT 4}}_{\text{OVARIES}}$

FUNDAMENTALS OF OBSTETRICS

I. CAPACITATION :



- ability of sperm to fertilize ova
- Occurs in Female Reproductive tract
- Begins in cervix, majority occurs in Fallopian tube
- After capacitation, sperms become Hypermotile

- Sperms become motile in → Caudal End of Epididymis
- sperms become Hypermotile in → Fallopian Tube

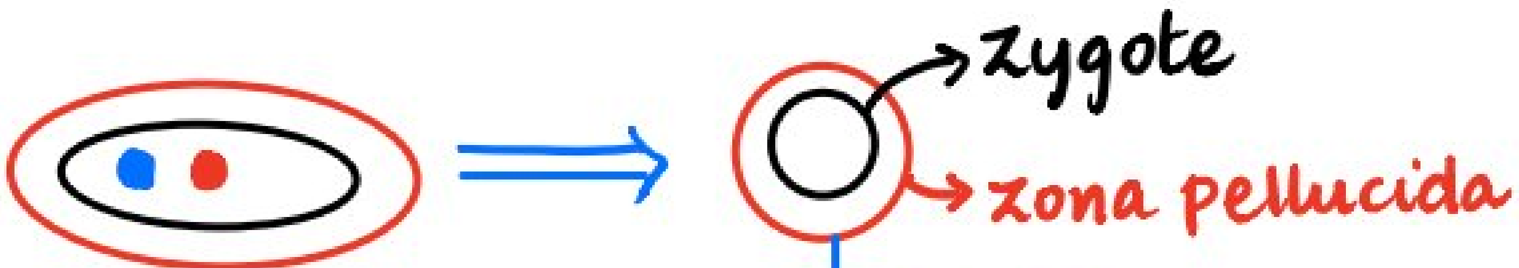
- Within 30mins → sperms reach Fallopian tube

ACROSOMAL REACTION

- Attaching of sperm to zona pellucida ; facilitated by certain receptors, they are ZP1, ZP-2, ZP-3

- Now it penetrates zona pellucida and reaches cortex of 2° oocyte
↓
Enzymes are released from cortex
↓
K/a 3. CORTICAL REACTION - ZP2 and Calcium ions are needed.
↓
4. ZONA REACTION : enzymes which are released, they make ZONA PELLUCIDA impermeable by other sperms.

function of zona pellucid is to prevent POLYSPERMY



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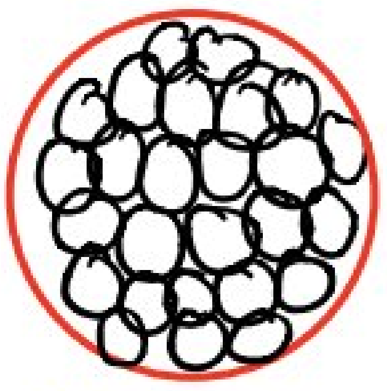
Fertilization
↓
occurs in Ampulla of Fallopian tube

20-30hr
2 cell stage
↓
4 cell stage
↓
8 celled Stage
↓
16 cell Stage : ZYGOTE
↓
K/a MORULA



- For 3 days after fertilization → Zygote remains in Fallopian tube

In Fallopian tube : 3 types of cells are there
① Columnar cells
② Secretory cells
③ PEG cells → Function is not known

- Nutrition for early zygote is provided by secretory cells of Fallopian tube

- For 3 days after fertilization zygote remains in Fallopian tube with help of 2 Factors -

① Peristalsis in tube
② Movement of cilia

} these 2 factors push ZYGOTE towards uterine cavity

- this explains if progesterone is used it can lead to ECTOPIC Pregnancy, since progesterone causes smooth muscle relaxation → leads to less peristalsis.

↓

B/w Cu and Progesterone IUCD's, chances of Ectopic pregnancy will be more with progesterone containing IUCD

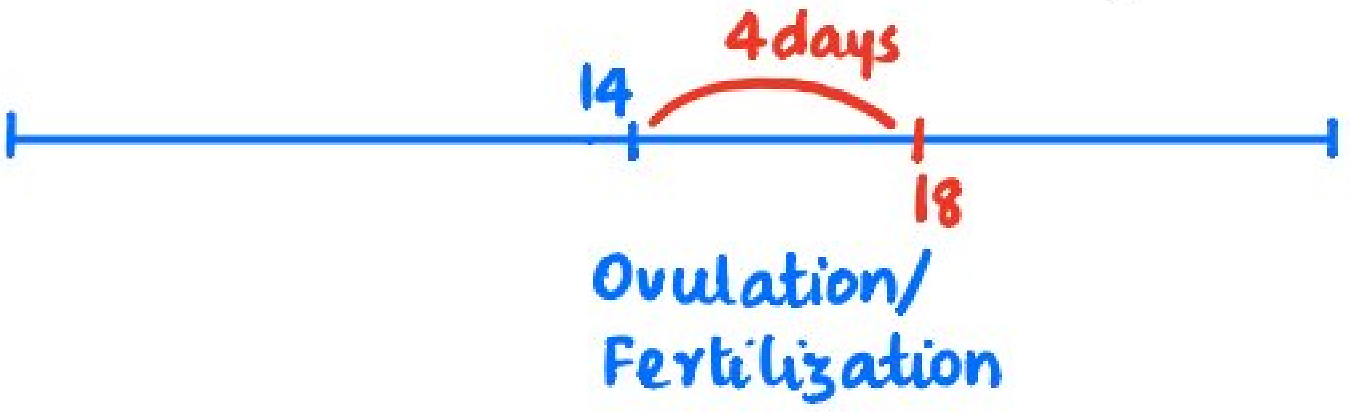
* If any tubal surgery was done, if after tubal surgery patient conceives → because in Tubal pregnancy adhesions would be formed → These adhesions prevents movement of zygote from the tube to uterine cavity → leads to Ectopic pregnancy (anything which is preventing movement of zygote towards uterine cavity leads to Ectopic pregnancy)

Q.

4th day : After fertilization / Day 18th of Menstrual cycle

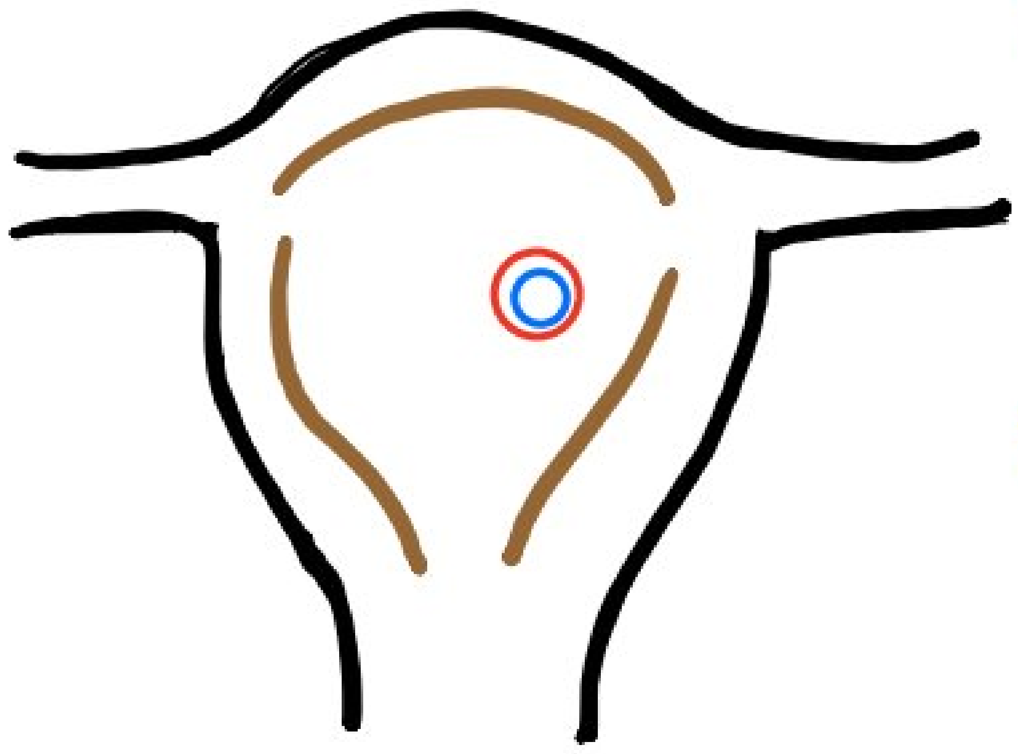
↳ MORULA enters uterine cavity

Menstrual cycle :



In all females * Length of cycle is 28 days * ovulation occurs on Day 14th

* Ovulation and Fertilization occurs on same day



* Day 5 after fertilization → ZONA PELLUCIDA is lost

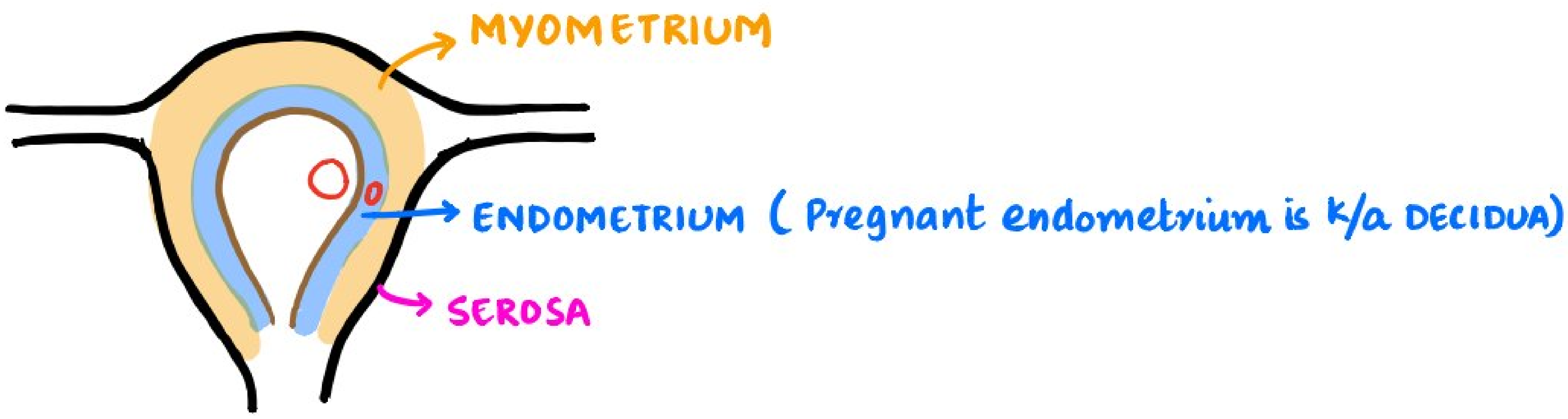
↓

K/A ZONA HATCHING

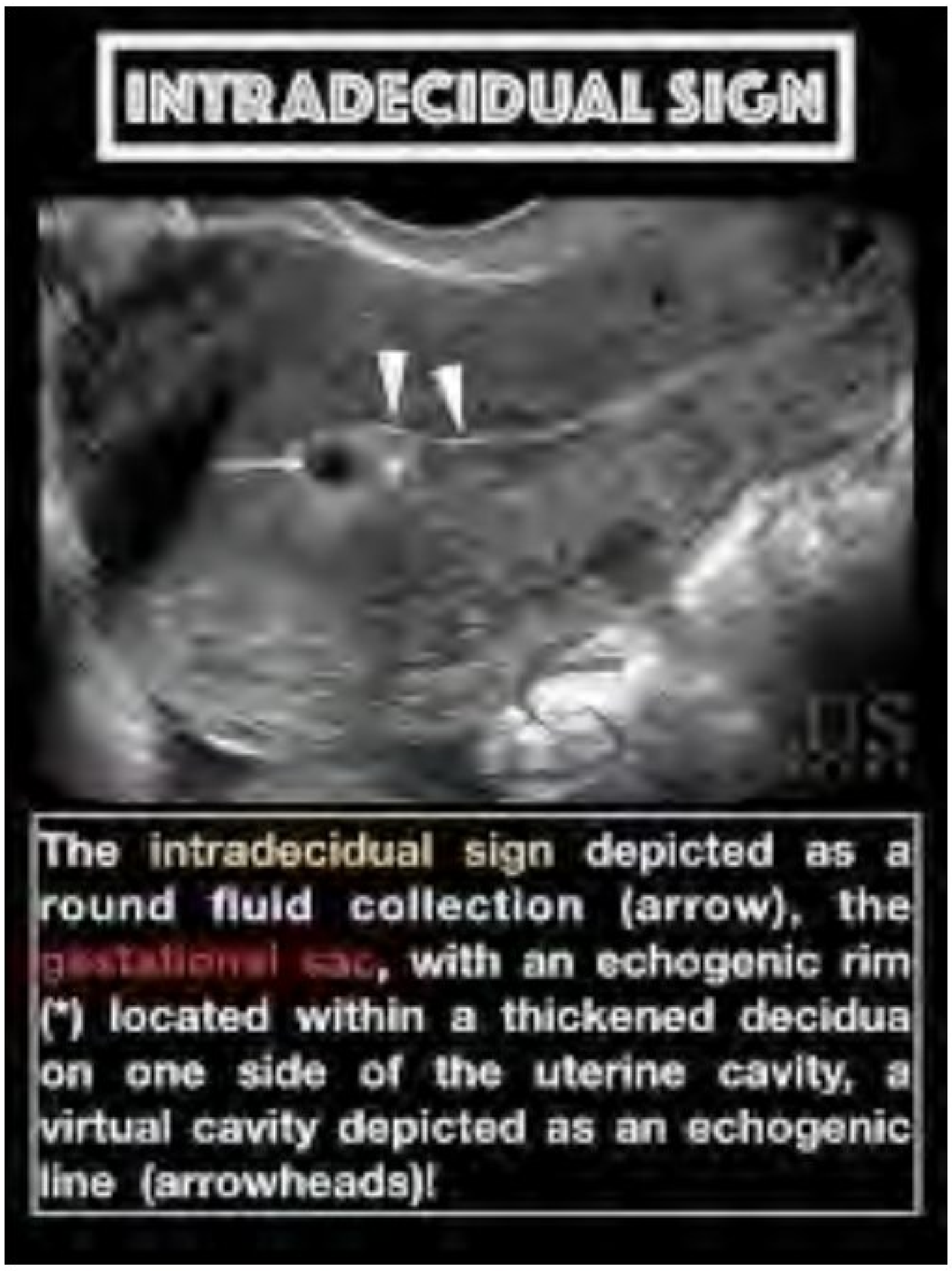
* Fluid enters morula → BLASTOCYST

↓

attaches to endometrium of uterus this is k/a IMPLANTATION

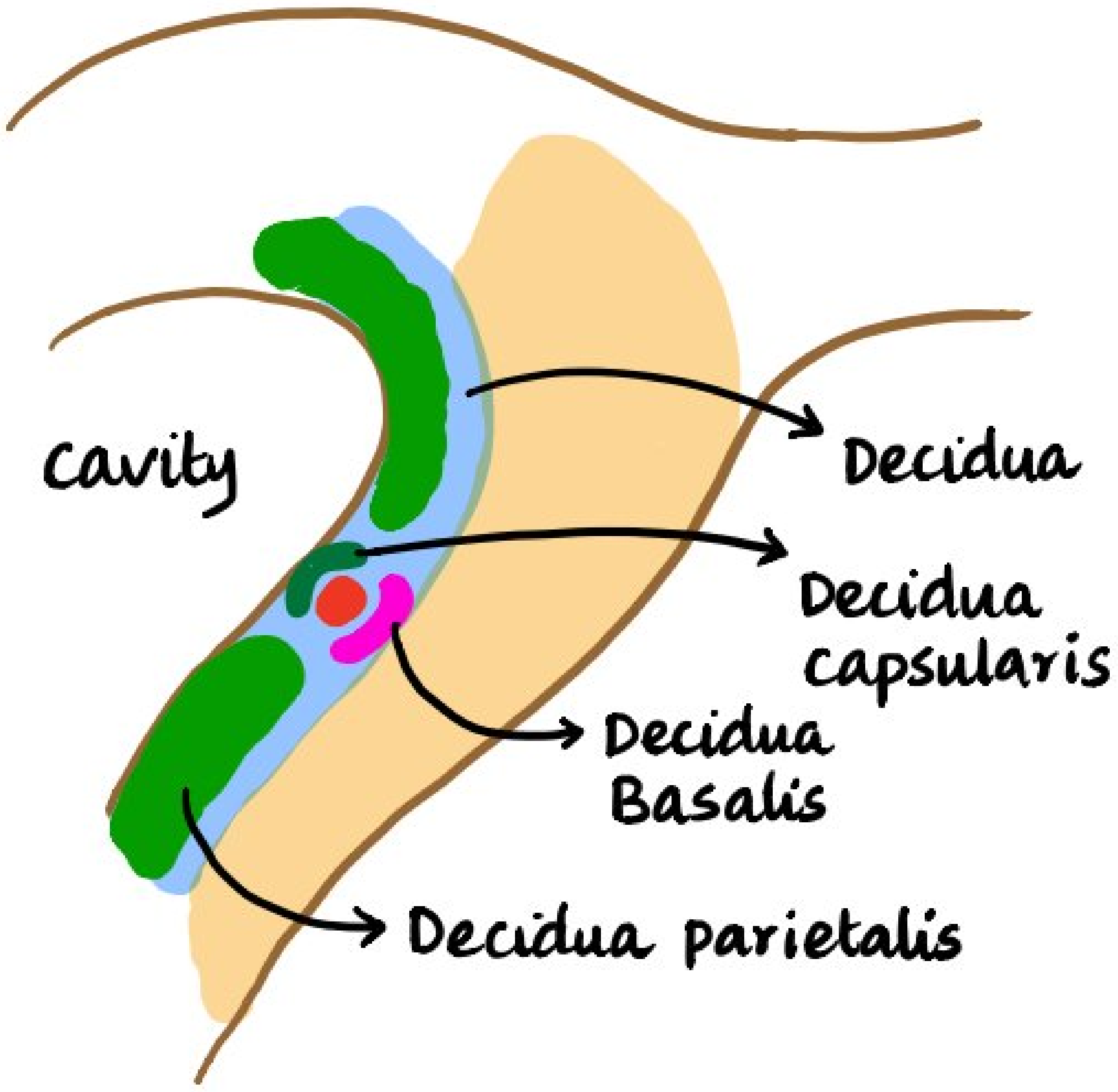


INTRA DECIDUAL SIGN
(1st Sign of Pregnancy on USG)



INTRADECIDUAL SIGN

The intradecidual sign depicted as a round fluid collection (arrow), the gestational sac, with an echogenic rim (*) located within a thickened decidua on one side of the uterine cavity, a virtual cavity depicted as an echogenic line (arrowheads)!



IMPORTANT POINTS ON IMPLANTATION

- Implantation occurs in the form of BLASTOCYST
- Begins by 6 days after fertilization (Day 20 of cycle)
- Implantation Window → Day 20 - Day 21
- Completed by 10 days after fertilization
- Most common site : upper posterior part of uterus

PSEUDOGESTATIONAL SAC

- centrally located
- Thickened decidua
- s/i ECTOPIC Pregnancy

- Implantation is ECCENTRIC (only on one side)

unequal growth of uterus

K/a PISKACEK Sign

True Gestational sac is eccentrically located

- Type of implantation is Interstitial Implantation
- Sign on USG → Intradecidual sign

* HARTMAN Sign/ Placental sign : Bleeding at time of Implantation

* Phases of Implantation →

Phase	Main transmitter
Apposition	Selectins
Adhesions	Integrins
Invasion	Metalloproteinase

* Thickness of Endometrium at time of Implantation is 10-12 mm

Thickness of Endometrium after menses is 0.5 mm

at ovulation is 2-3 mm

in Secretory phase - 5-6 mm

* After Implantation : ENDOMETRIUM ↓

K/A DECIDUA

1. DECIDUA BASALIS

* part of decidua which separates Blastocyst from myometrium

* Function :

i) Site for formation of Future placenta

ii) Forms the maternal side of placenta

2. DECIDUA CAPSULARIS

↓

* which separates Blastocyst from Uterine cavity

3. DECIDUA PARIETALIS

* Rest of decidua lining uterus

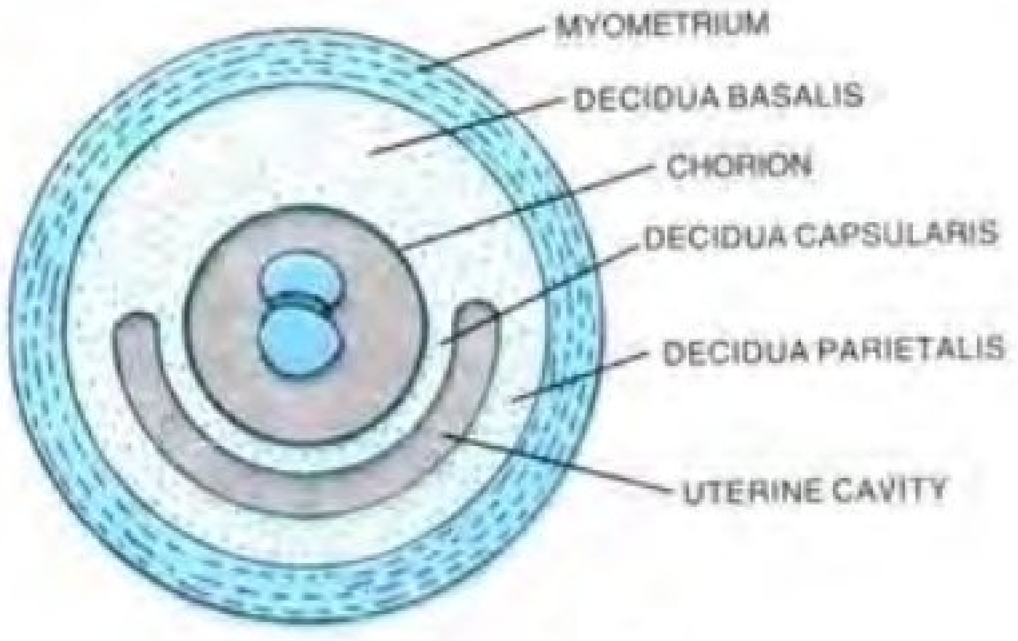
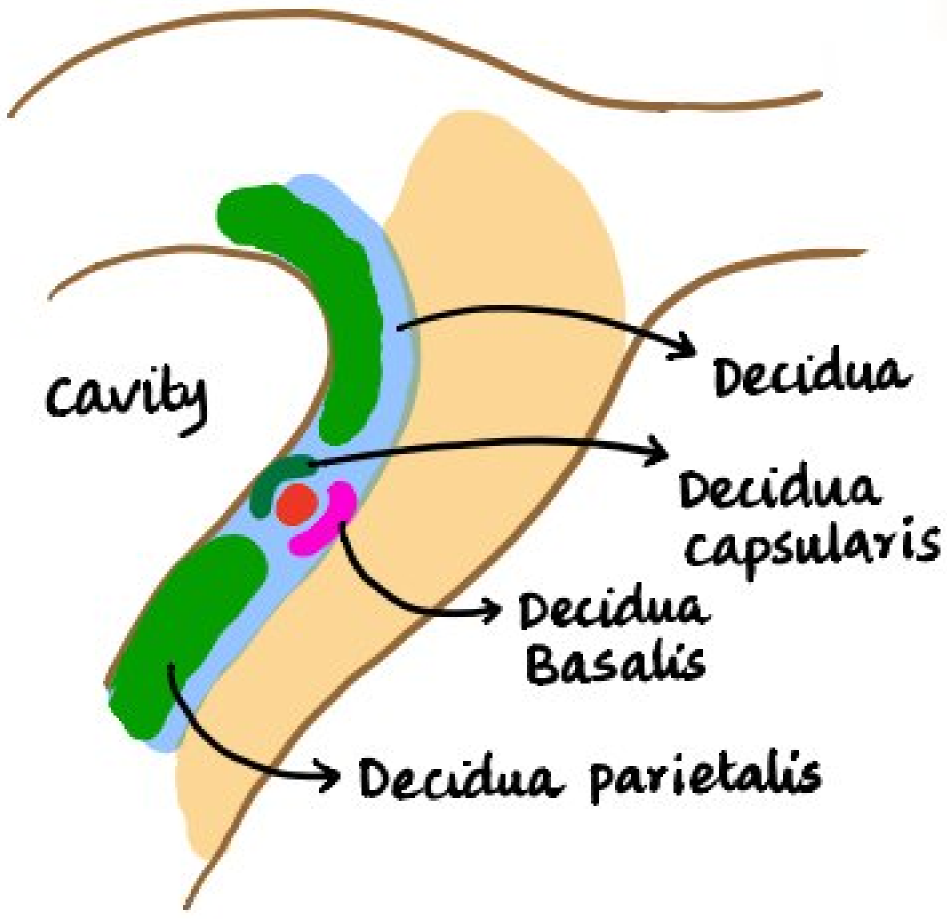
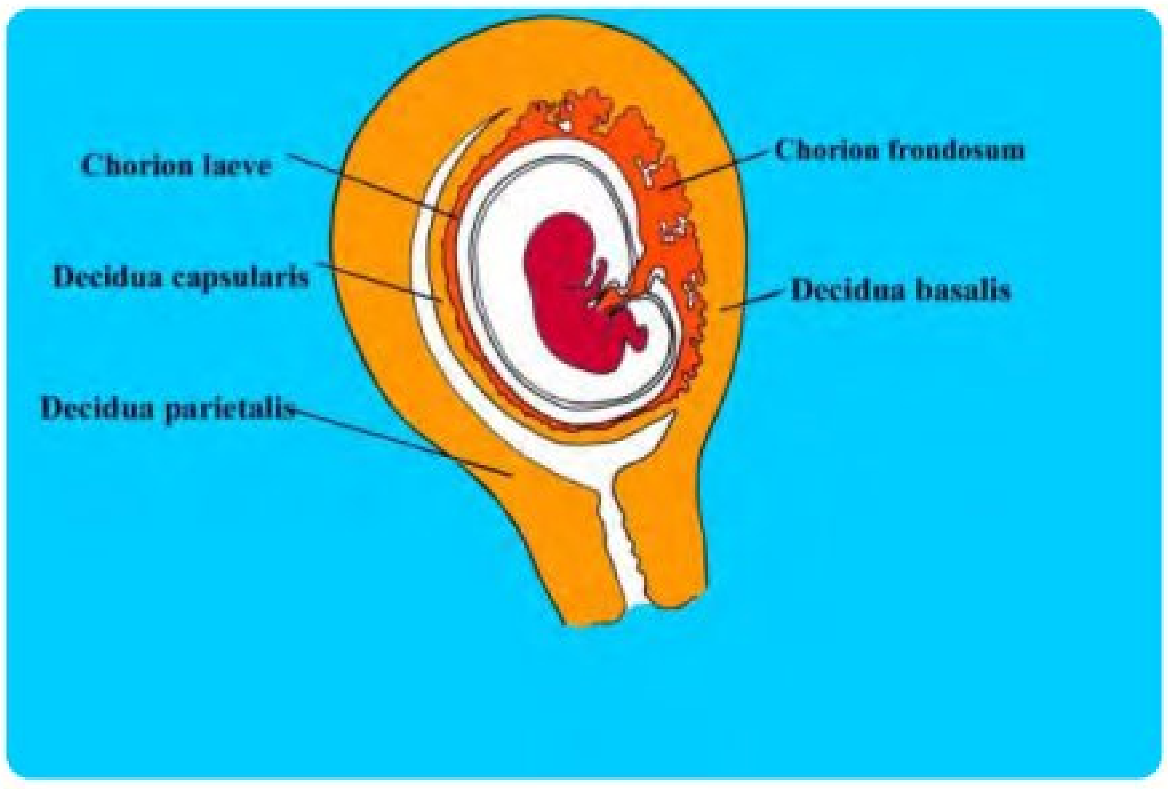


Fig. 3.30. Subdivisions of decidua.





* As Blastocyst grows → it occupies uterine cavity and decidua capsularis and parietalis come close to each other

Q. Decidua Capsularis and Decidua parietalis fuse at 14 - 16 wks of pregnancy

Q. When is uterine cavity obliterated → 14 - 16 wks of pregnancy

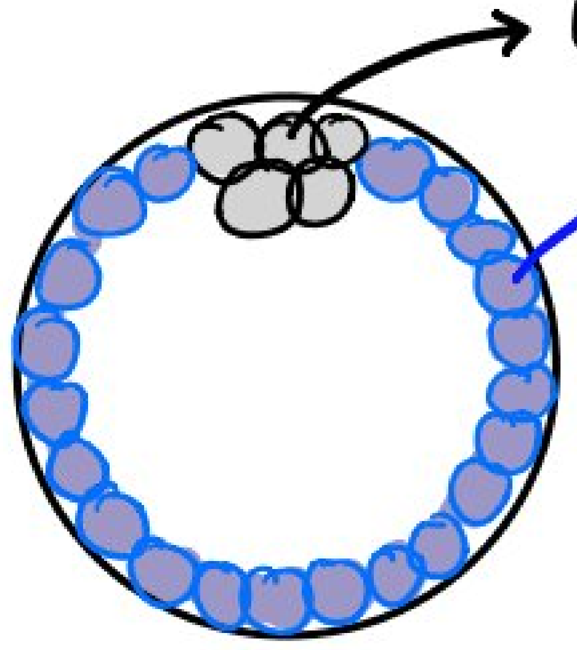
TWINS (2 ova fertilized by 2 sperms)

In same cycle
↓
SUPERFECUNDATION
s/i Humans

In 2 different cycles
↓
SUPERFETATION
NOT S/i HUMANS

Q. Theoretically Superfetation is possible till 14 - 16 wks

BLASTOCYST - 58 cells



→ Inner cell mass (5 cells) → forms entire fetal part

→ TROPHOBLAST (53 cells)
8 days after fertilization

Cytotrophoblast

Synctiotrophoblast
↓
Hormone Factory of Placenta

Near decidua basalis
↓
forms vili like structures
↓
K/a CHORION FRONDOSUM
↓
forms fetal side of placenta

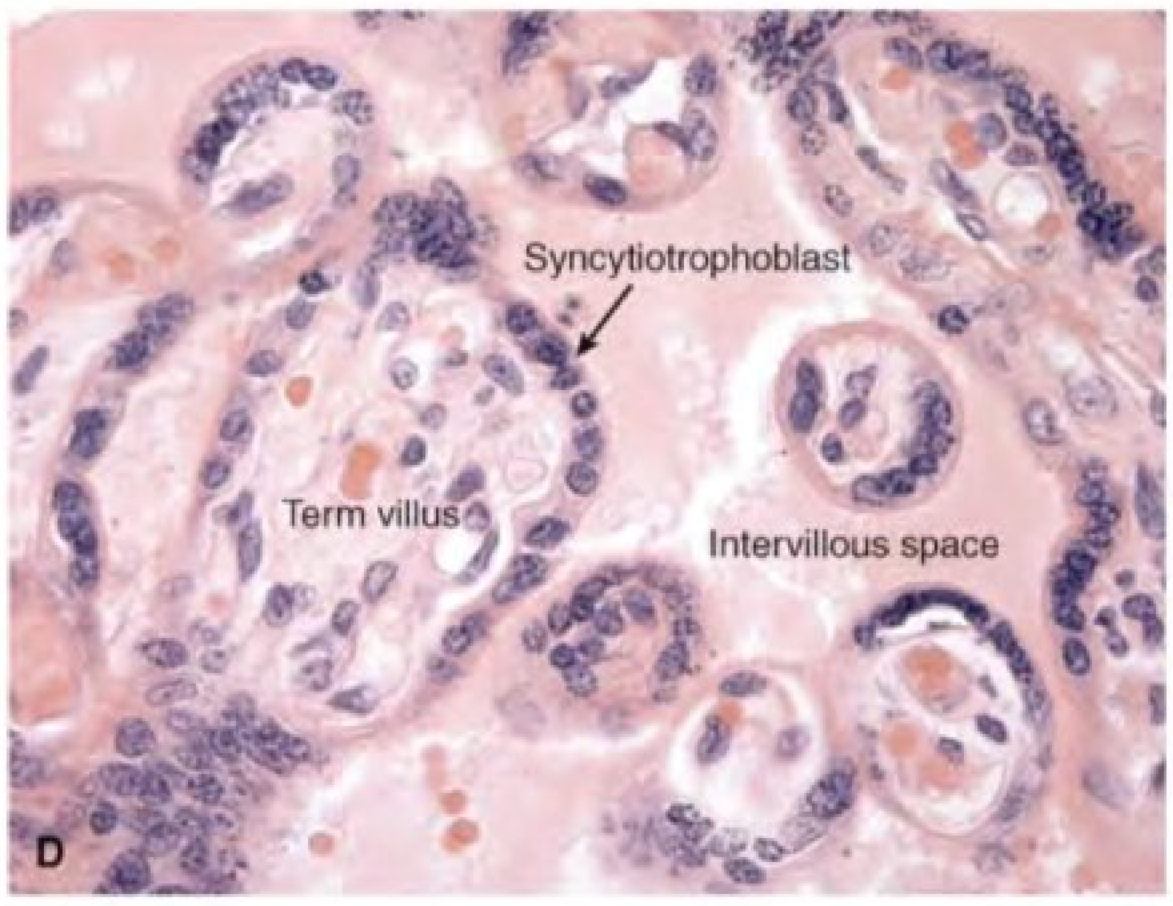
Rest of chorion Remains smooth
↓
K/a CHORION LAEVE
↓
CHORION

prevents PIH by converting maternal arteries from High Resistance to low Resistance vessels
↓
K/a TROPHOBLASTIC INVASION
↓
occurs in 2 phases
1st phase begins - 12 wks
2nd phase begins - 16 wks

* carried by Extra villous Cytotrophoblast

* Controlled by NK Cells of decidua

"uteroplacental blood flow is maintained"

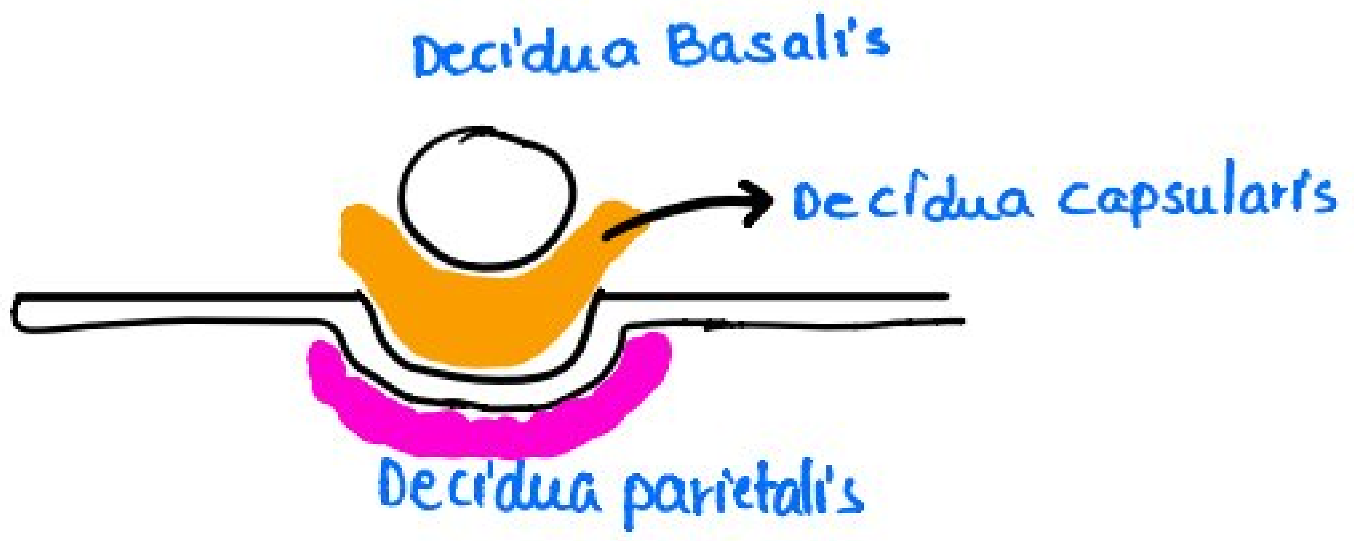


If Trophoblastic invasion is ABSENT
(uteroplacental Insufficiency)

PIH

IUGR

PLACENTA ACCRETA



"DOUBLE DECIDUAL RING SIGN"
(seen only in Intrauterine pregnancy)

1st Ring - Decidua capsularis

2nd Ring - Decidua parietalis

Placenta and Amniotic Fluid

DISCOID	DECIDUATE	HEMOCHORIAL
↓	↓	↓
DISC Like Shape	Sheds off after delivery	Lies directly in contact with maternal blood

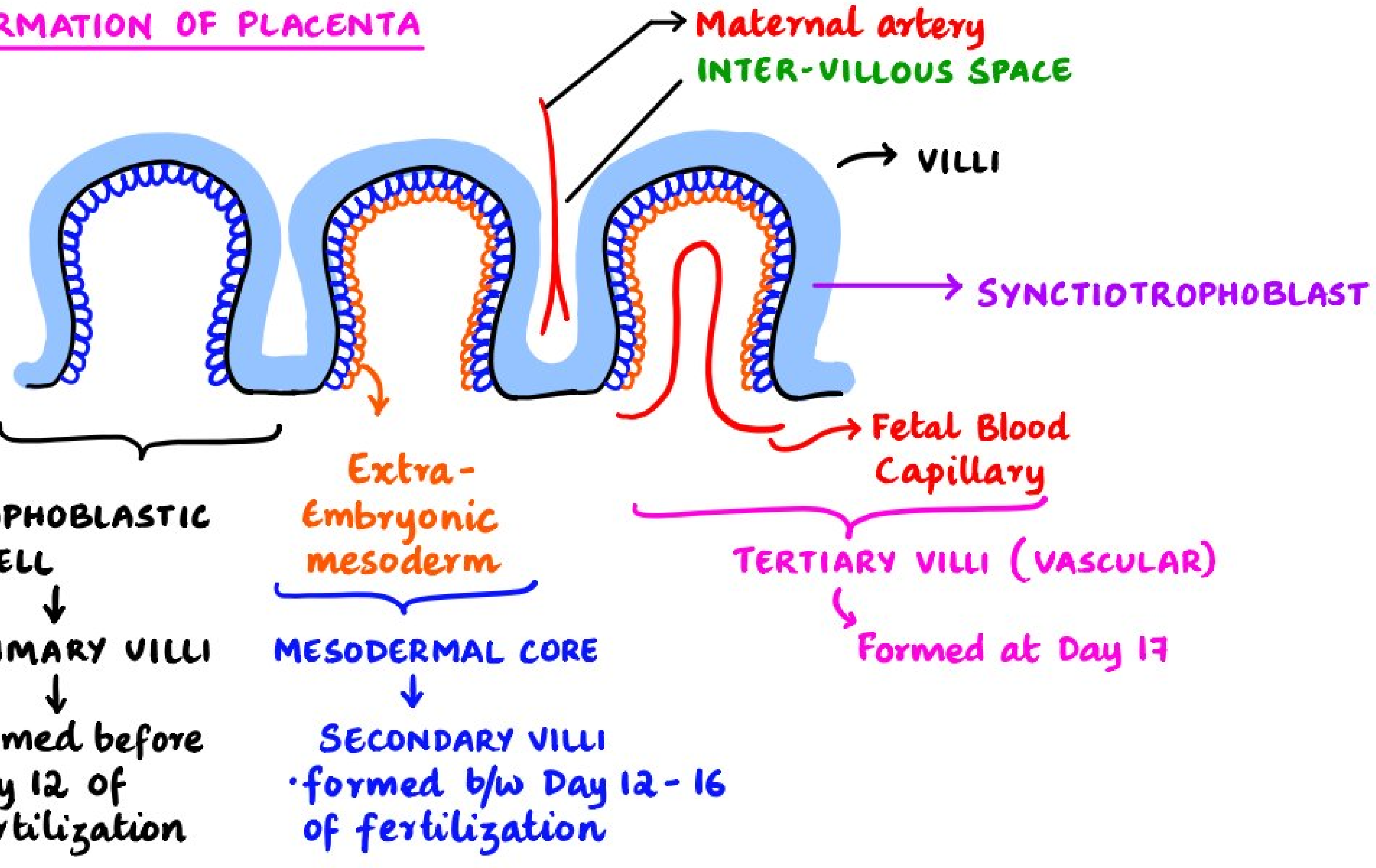
- Weight of Placenta at TERM : 500g
- Ratio of weight of Placenta : Fetus = 1:6 at term

Q. Weight of Placenta equals the weight of fetus at 17wks

NAMED STRUCTURE	ORGAN SEEN
HOFFBAEUR CELLS	PLACENTA
PEG CELLS	FALLOPIAN TUBE
LANGHAN CELLS	CYTOTRHOPHOBLAST
NITABUCH LAYER	FIBRINOID DEGENERATION which is present between CHORION and DECIDUA

* ZIKA VIRUS CAN INFECT HOFFBAEUR CELLS

FORMATION OF PLACENTA



TROPHOBLASTIC SHELL
↓
PRIMARY VILLI
↓
formed before Day 12 of fertilization

MESODERMAL CORE
↓
SECONDARY VILLI
- formed b/w Day 12 - 16 of fertilization

TERTIARY VILLI (VASCULAR)
Formed at Day 17

Q.
Fetal circulation established in placenta on Day 17
Maternal Blood capillaries open into intervillous space on Day 15

Q. (AIIMS)

Maternal Blood in Intervillous space is separated from fetal blood in villi by

↓

(From outside to inside)

1. Synctiotrophoblast
2. Cytotrophoblast
3. Extra Embryonic Mesoderm
4. Fetal Capillary Endothelium

} PLACENTAL BARRIER/ MEMBRANE

Placental circulation

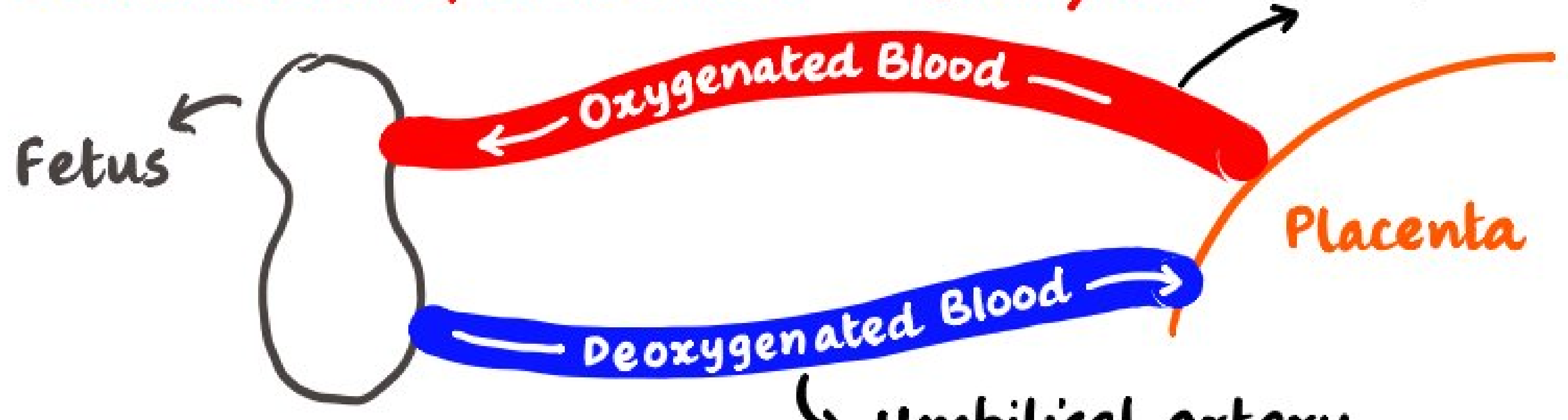
UTEROPLACENTAL (In Intervillous space) ↓

- Established on Day 15
- No. of spiral arteries which open in intervillous space - 120
- O_2 saturation in intervillous space ⟶ 65 - 75%
- Partial pressure in intervillous space ⟶ 35 - 45mm Hg
- Uteroplacental Blood flow at 36 wks - 500 - 750 ml/min

Fetoplacental (In villi) ↓

- Established by Day 17 after fertilization
- Fetoplacental circulation at term - 400 ml/min
- Fetal Blood flow at term - 125ml/Kg
- Rate of delivery of O_2 to fetus - 8ml/Kg/min

Q. Uterine Blood flow at term - 750ml/min



UMBILICAL VEIN:

- O_2 Sat = 70-80%
- Remnant: Ligamentum teres

Umbilical artery

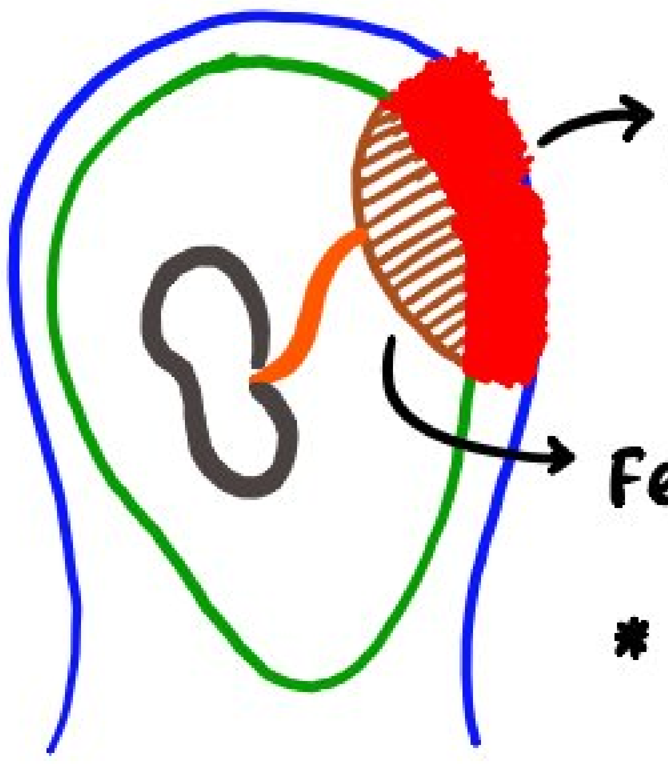
- O_2 saturation : 50-60%
- Remnant : Medial Umbilical ligament

Q.

Median Umbilical ligament = Remnant of URACHUS

Lateral Umbilical ligament = Remnant of Inferior Epigastric Artery

ANATOMY OF PLACENTA



Maternal side : 1/5th of Placenta

Fetal Side : 4/5th Placenta

* But Both of them occupy same area on Uterine wall.

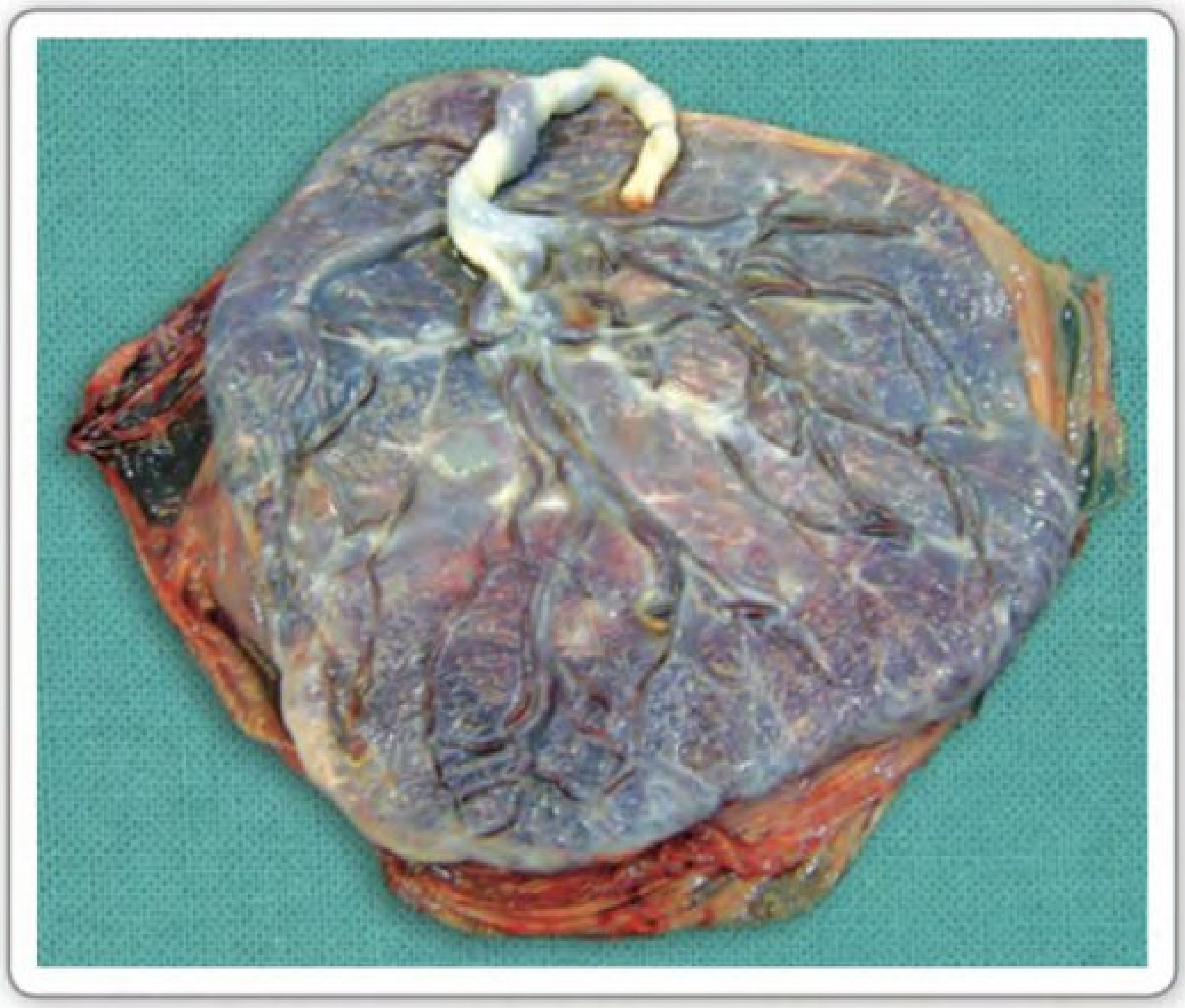


Fig. 3.2: Fetal surface of the placenta showing attachment of the umbilical cord with ramification of the umbilical vessels

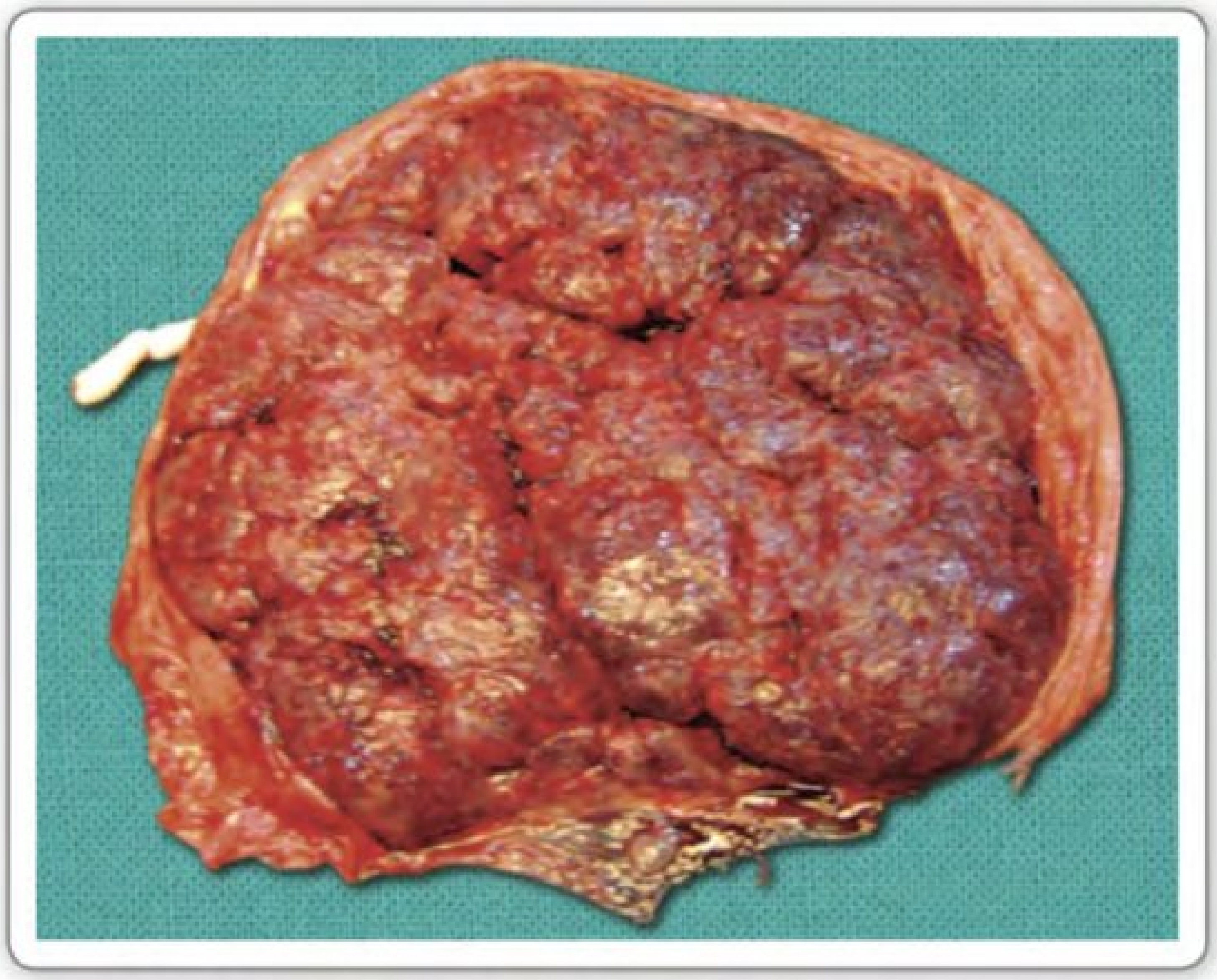


Fig. 3.3: Maternal surface of the placenta showing shaggy look with cotyledons limited by fissures

FETAL SIDE

- formed by CHORION FRONDOSUM

  2nd Best : CYTOTROPHOBLAST
  3rd Best : TROPHOBLAST

- It comprises of 4/5th of placenta
- Shiny, grey in colour
- Fetal membrane, umbilical cord are attached to this side

MATERNAL SIDE

- It is formed by DECIDUA BASALIS
- comprises of 1/5th of Placenta
- Dull Red in Colour
- It is divided into Polygonal areas ← aka LOBES

  further divided into lobules/ Cotyledons

IMPORTANT POINTS

- Functional unit of placenta – **COTYLEDON** (area which is supplied by main stem villi and all its branches)
- USG for localization of placenta – 3rd TM
- Thickness of placenta at term – Avg. 2·5cm (every wk, thickness of placenta increases by 1mm, so maximum in 40 wks, it can reach 40 mm or 4 cm)

PLACENTOMEGALY → Thickness >4cm

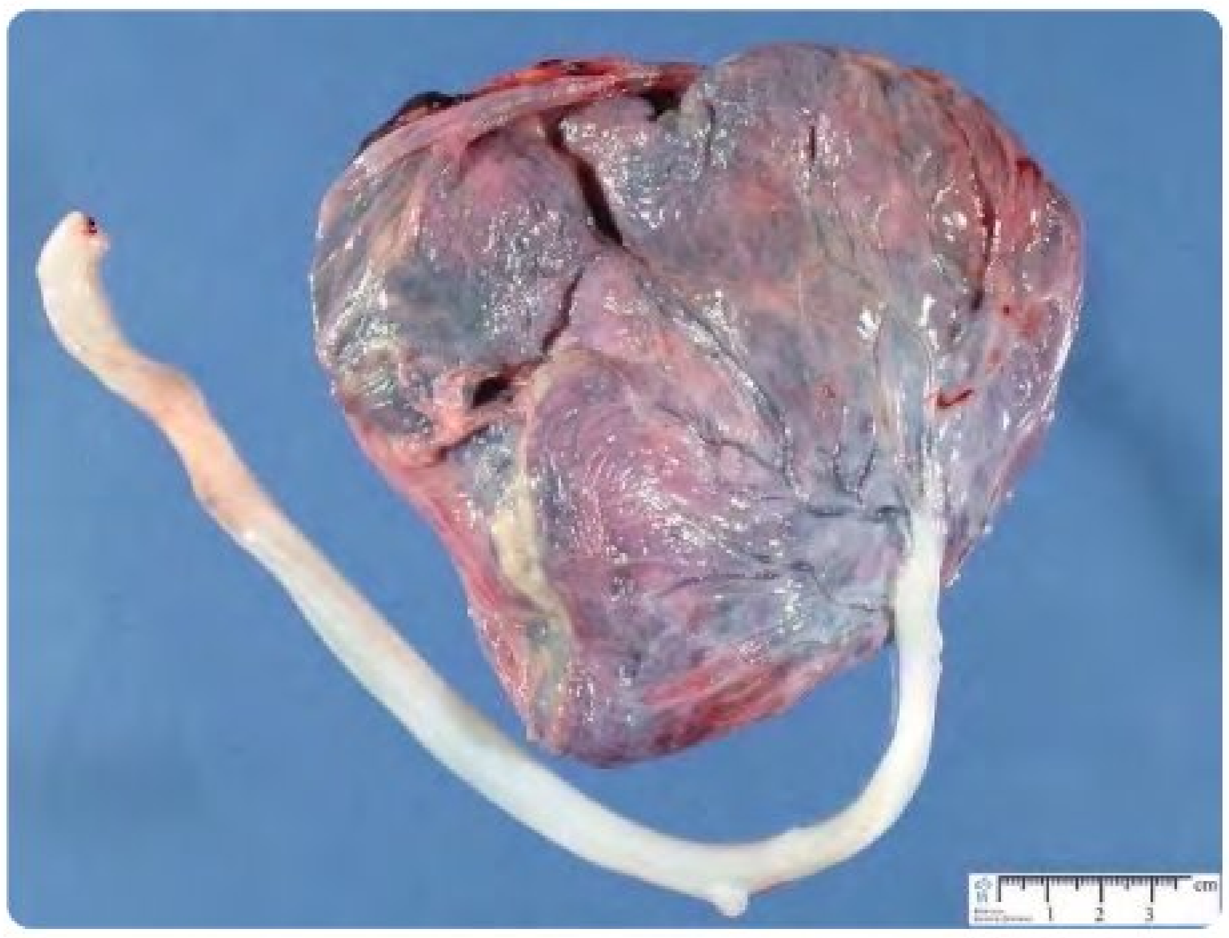
↓

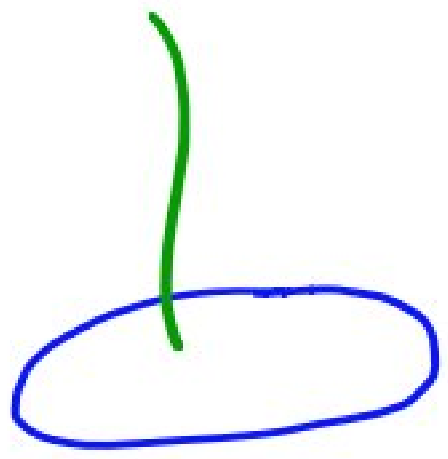
Causes

Maternal causes	Fetal causes
1. Diabetes	1. Hydrops fetalis
2. Severe anemia	2. Infection (Syphilis, TORCH)
3. α-Thalassemia	3. TRIPLOIDY

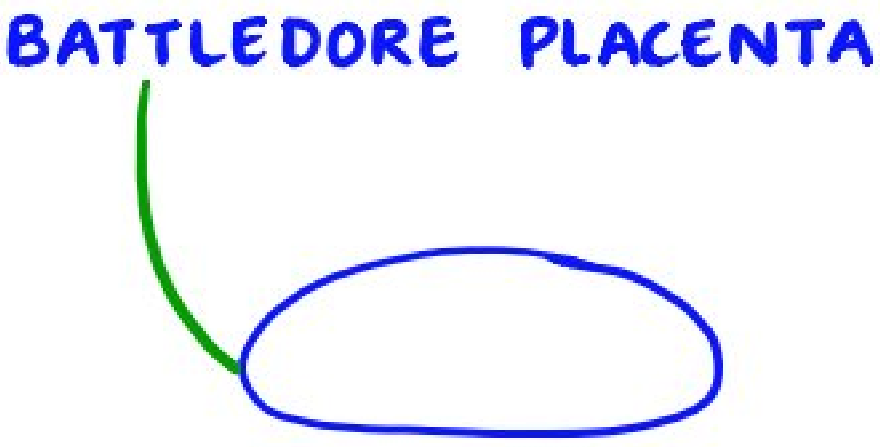
ANOMALIES OF PLACENTA

1. BATTLEDORE PLACENTA





Normal : central cord insertion



aka Marginal Insertion of cord

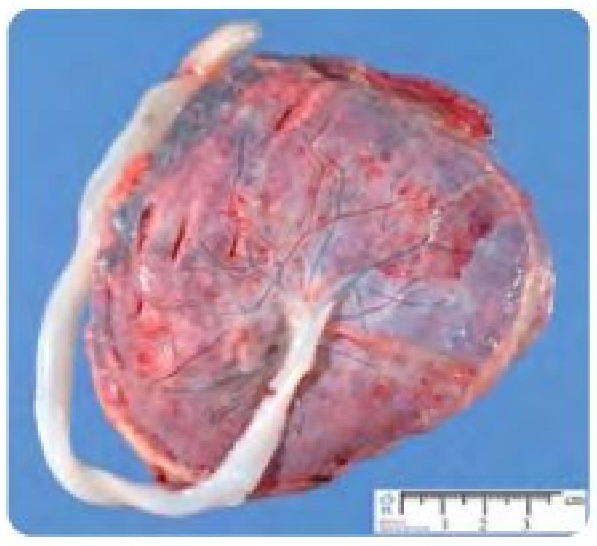
2. EXTRACHORIAL PLACENTA

- when fetal side is smaller than maternal side such that fetal side is surrounded by maternal side in the form of Ring

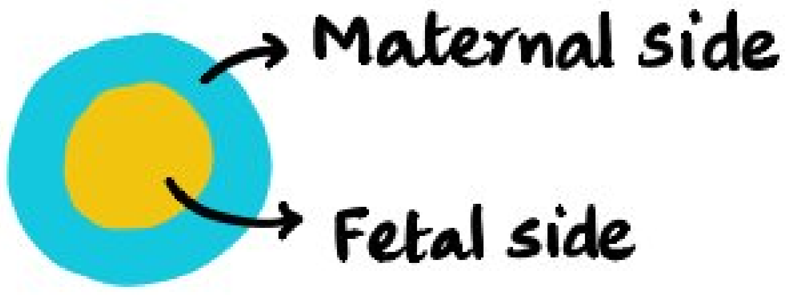
(Normally → Fetal side is equal to maternal side

Fetal side = CHORIONIC PLATE

Maternal side = DECIDUAL PLATE)

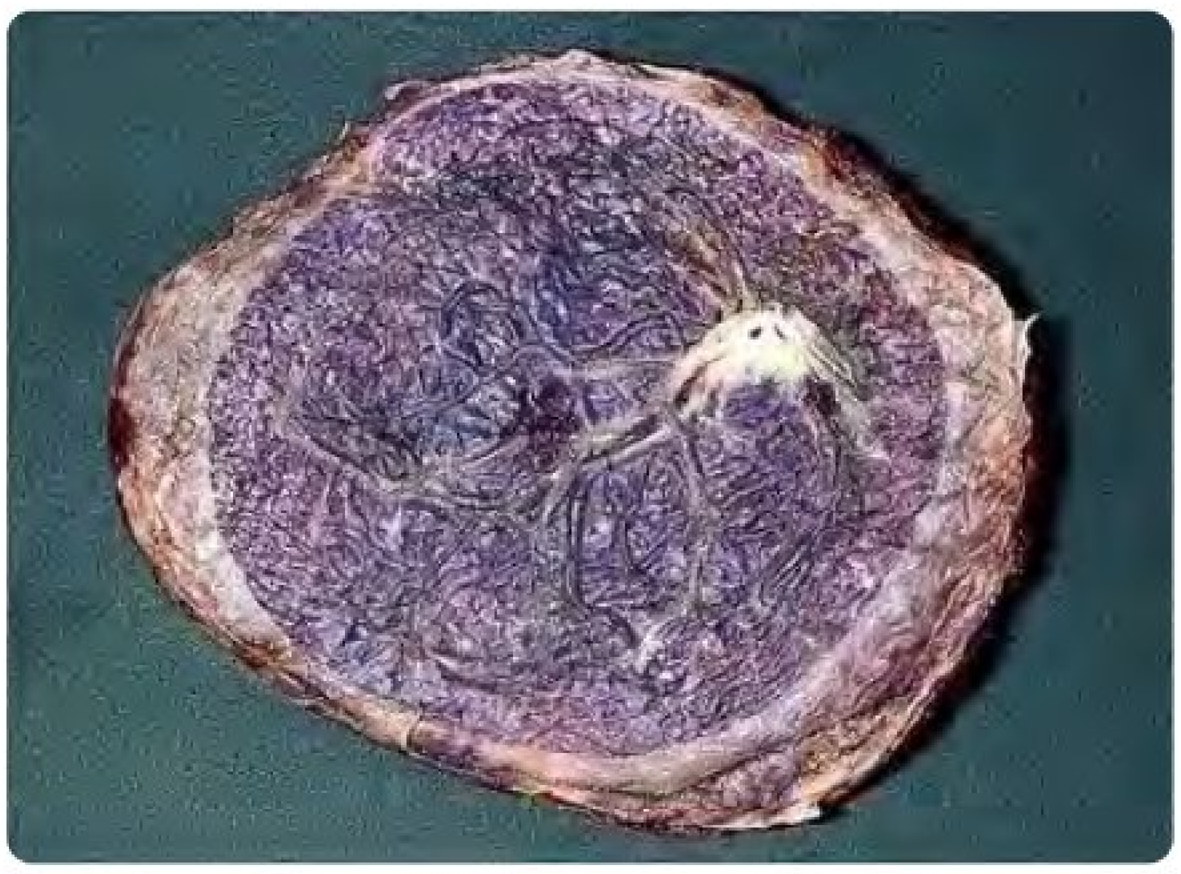


1. CIRCUMMARGINATE



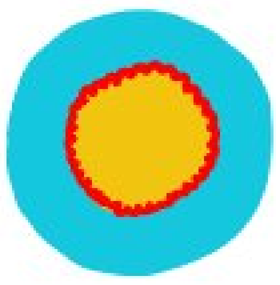
- fetal side is surrounded by Maternal side smoothly

↓



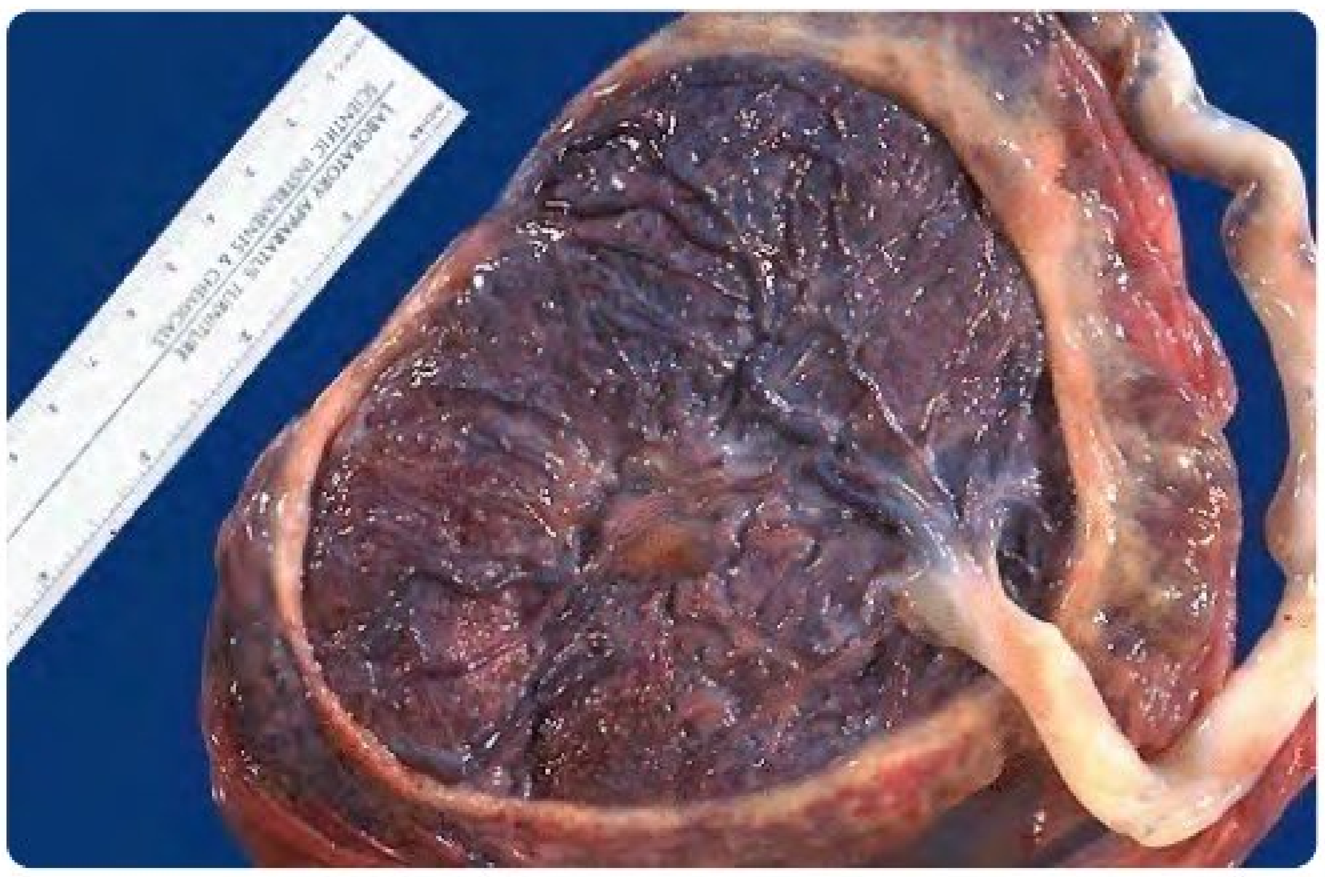
- fetal side and maternal are at same level

2. CIRCUMVALLATE



- fetal side is surrounded by maternal side but a valve like thickening is present.

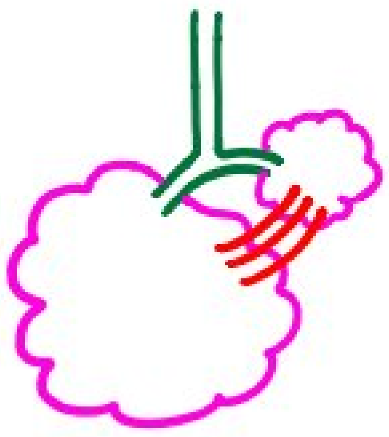
↓



- (fetal side will be little depressed)

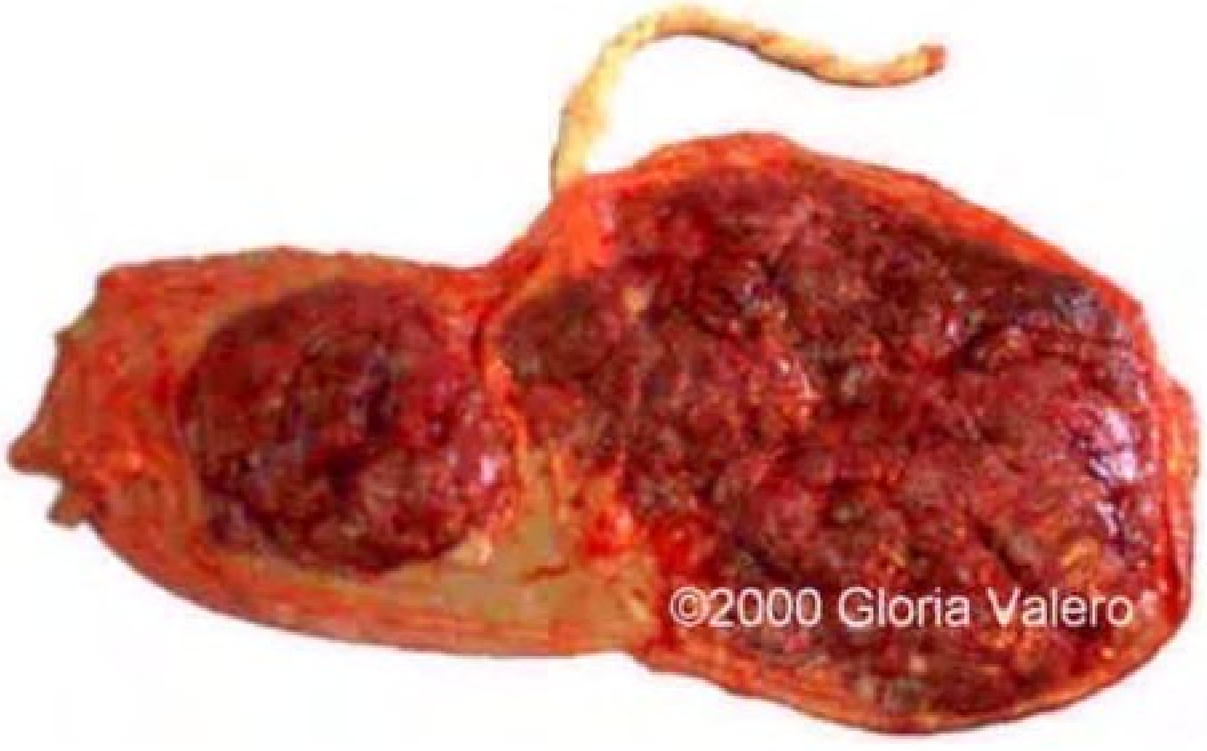
These 2 can lead to IUGR and APH

3.



i) Placenta succenturiata

- small parts detach from main placenta
- connecting blood vessels in b/w them ⊕



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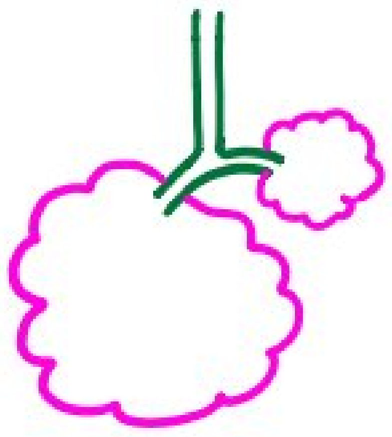
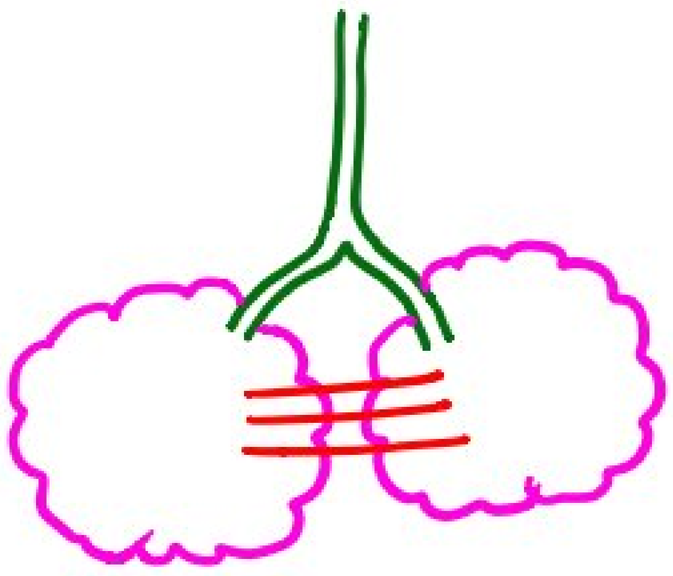
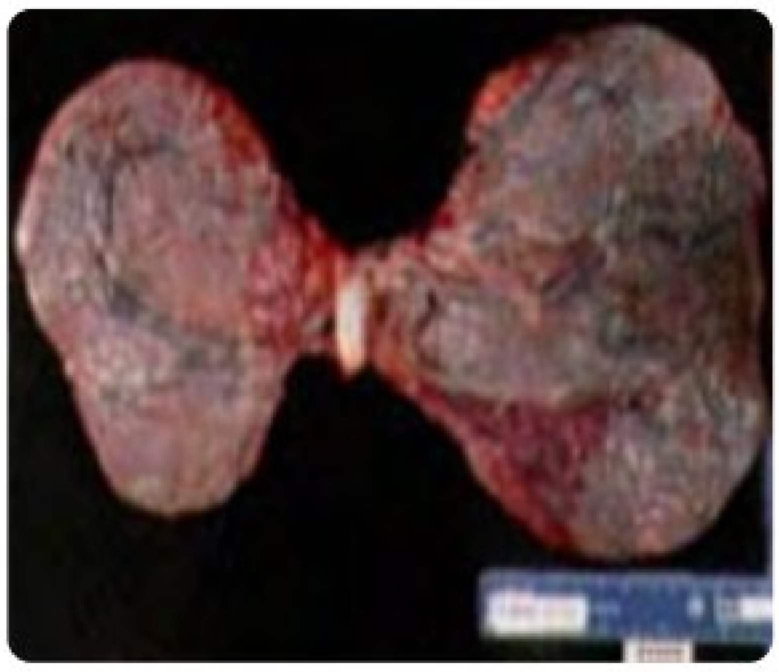


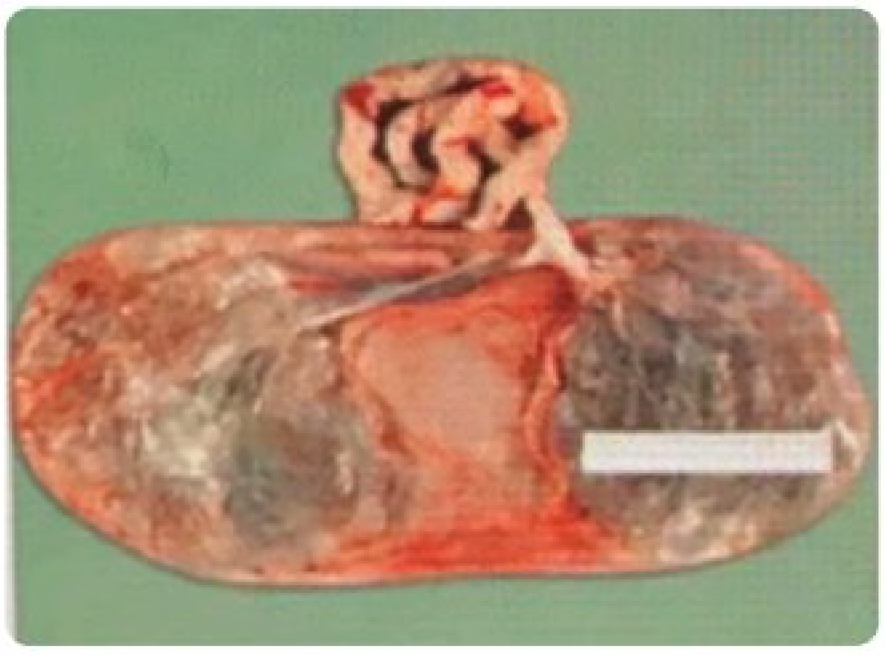
ii) PLACENTA SPURIA

- small part detaches from main placenta
- NO CONNECTING Blood vessels

iii) Placenta Bilobata / Placenta Biparite / PLACENTA DUPLEX







- 2 equal parts of placenta (separate)
- connecting blood vessels are seen

- All these 3 to PPH (2° PPH)

Mx: CURETTAGE

↳ when we do curettage in postpartum patient (can lead to ASHERMAN Syndrome)

↓ (MC) cause of ASHERMAN

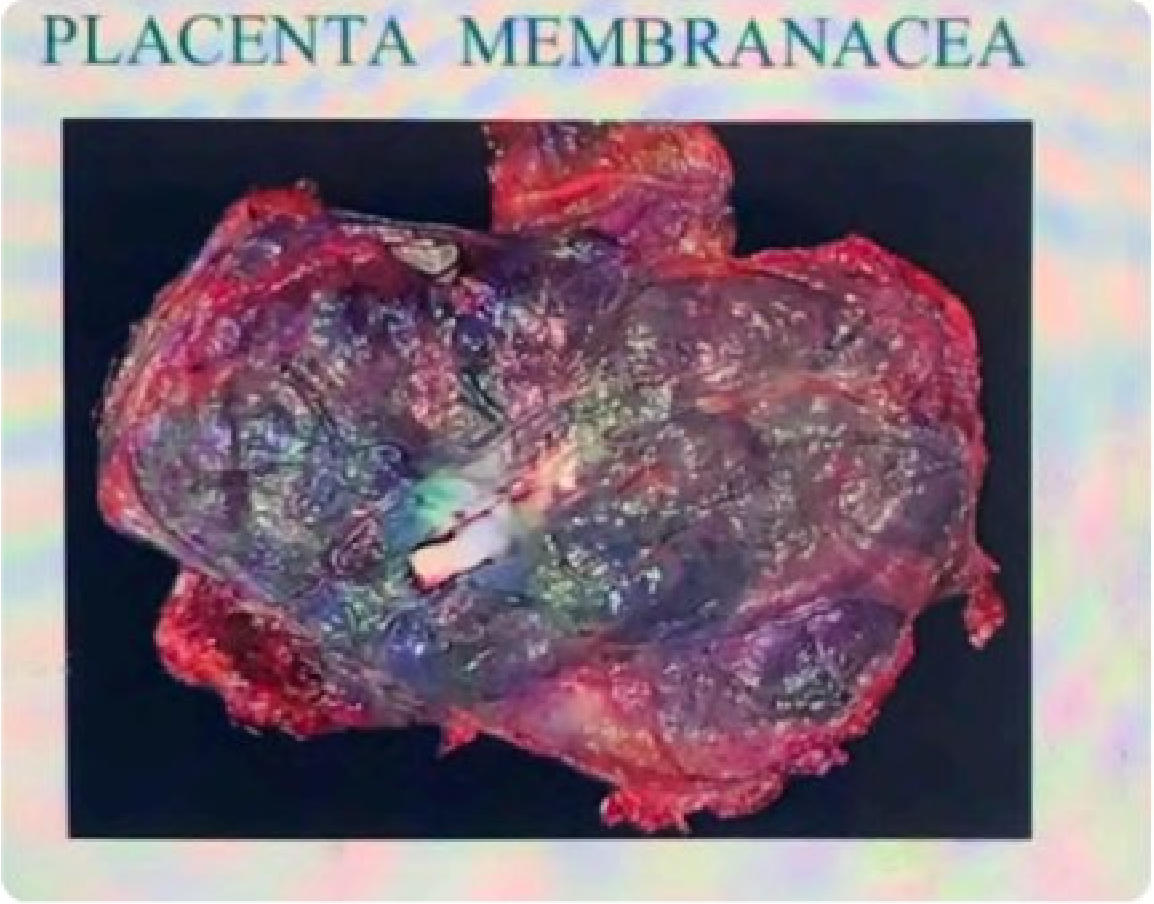
↓ Intra-uterine adhesions

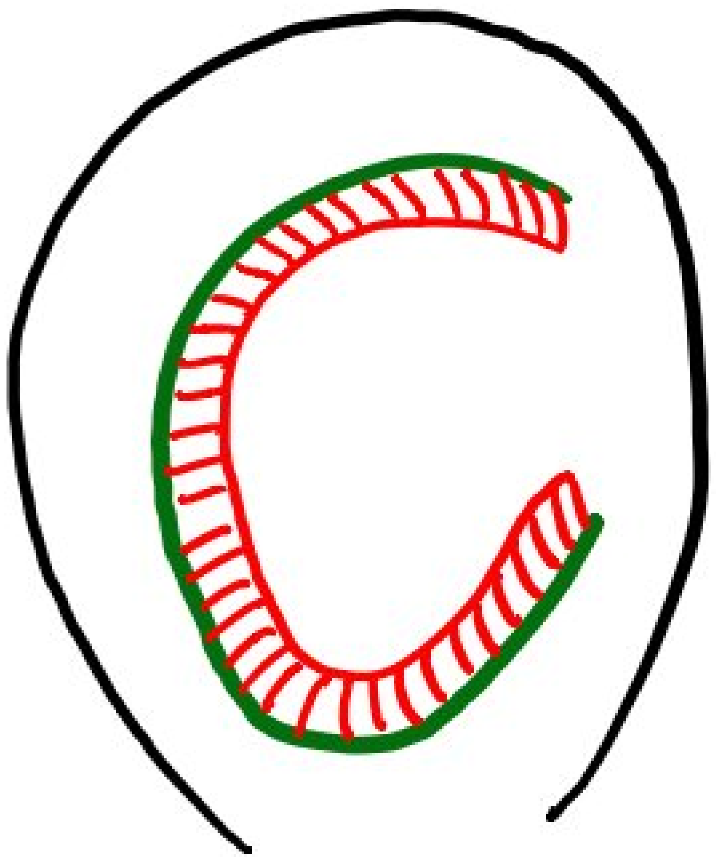
4. FENESTRATED PLACENTA

- central portion of a discoidal placenta is missing
- In some instances, there is an actual hole in placenta but more often the defect involves only villous tissue with chorionic plate mistakenly considered to indicate that a missing portion of placenta

5. PLACENTA MEMBRANACEA

- all of the fetal membrane are covered by functioning villi and the placenta develops as a thin membranous structure occupying the entire periphery of the chorion
- serious hemorrhage due to placenta praevia or placenta accreta

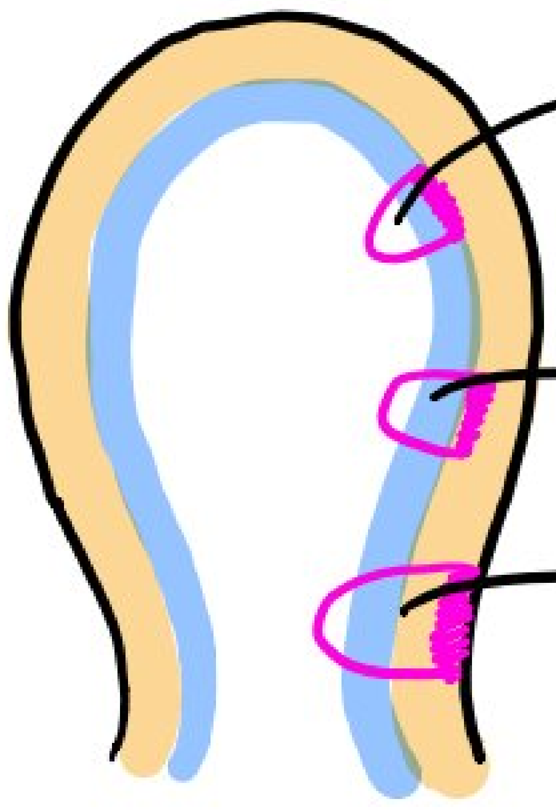




6. PLACENTA ACCRETA

***NITABUCH's Layer :**
- Layer of fibrinoid degeneration which is present b/w placenta and decidua
- It limits the penetration of BLASTOCYST

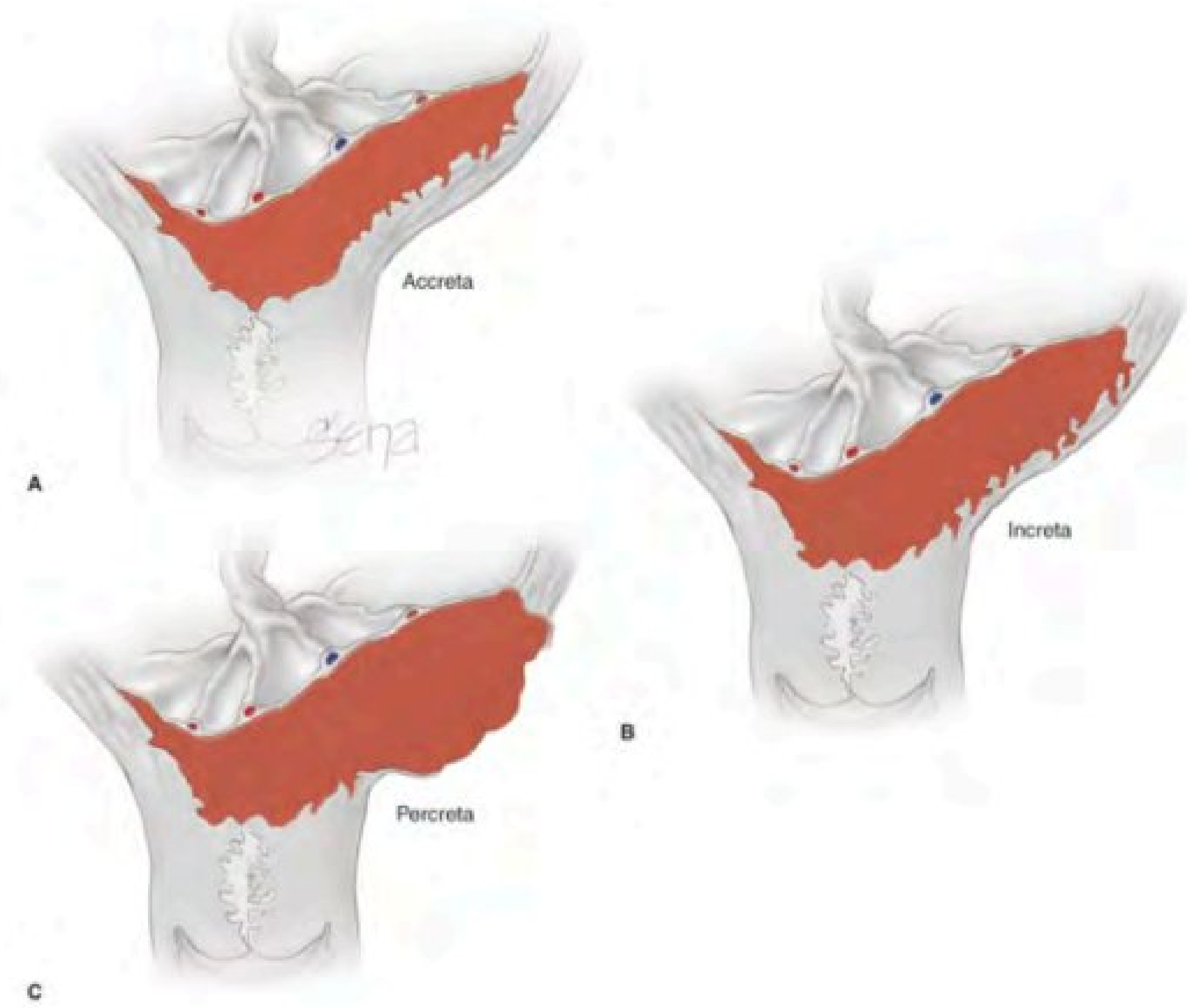
If NITABUCH's Layer is ABSENT : $\xrightarrow{\text{leads to}}$ **MORBIDLY ADHERENT PLACENTA**

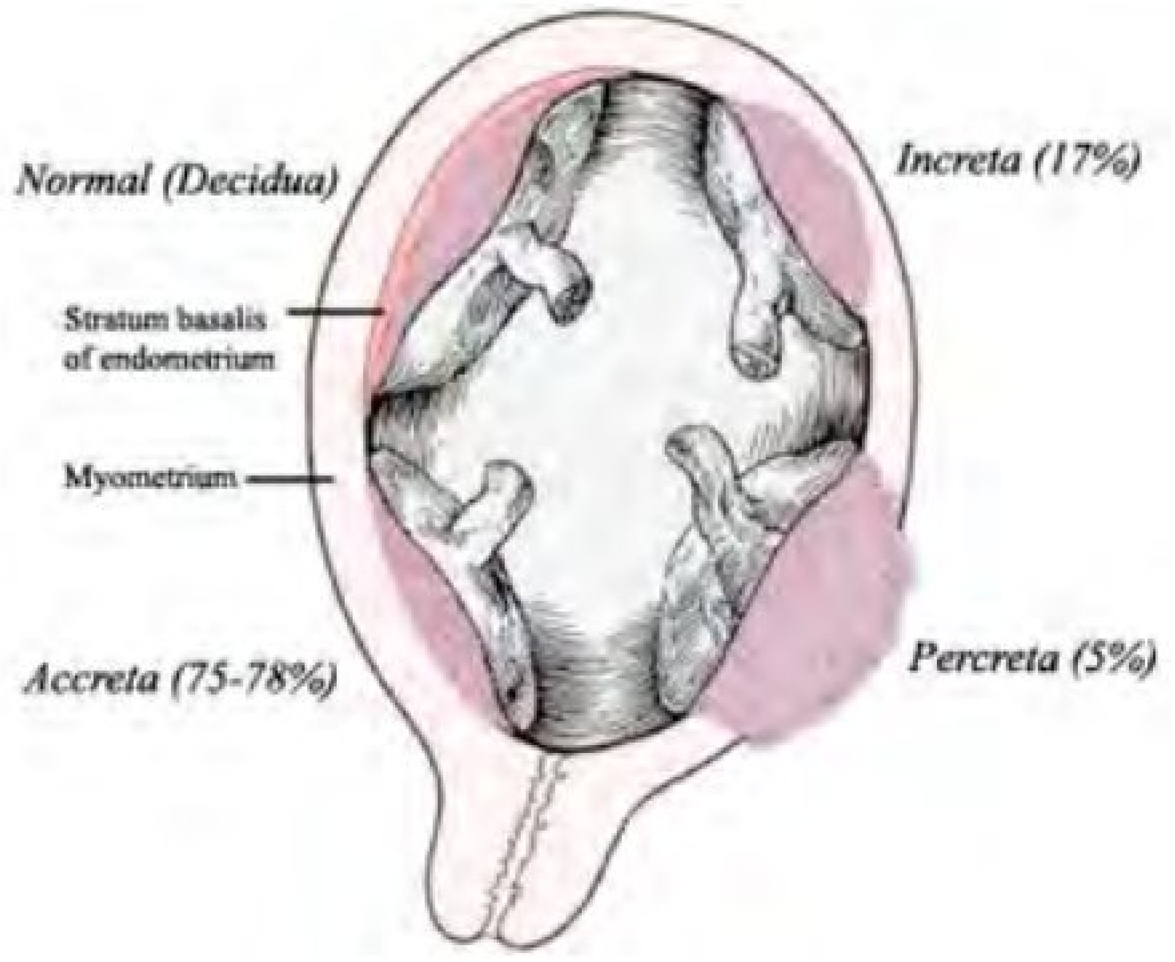


1. Placenta accreta - superficially attached to myometrium
2. Placenta increta - Deeply attached to myometrium
3. Placenta percreta - Chorionic villi attached to serosa

* in all these 3 conditions, there are 2 Pathologies

1. Nitabuch's layer is ABSENT
2. DECIDUA BASALIS is ABSENT





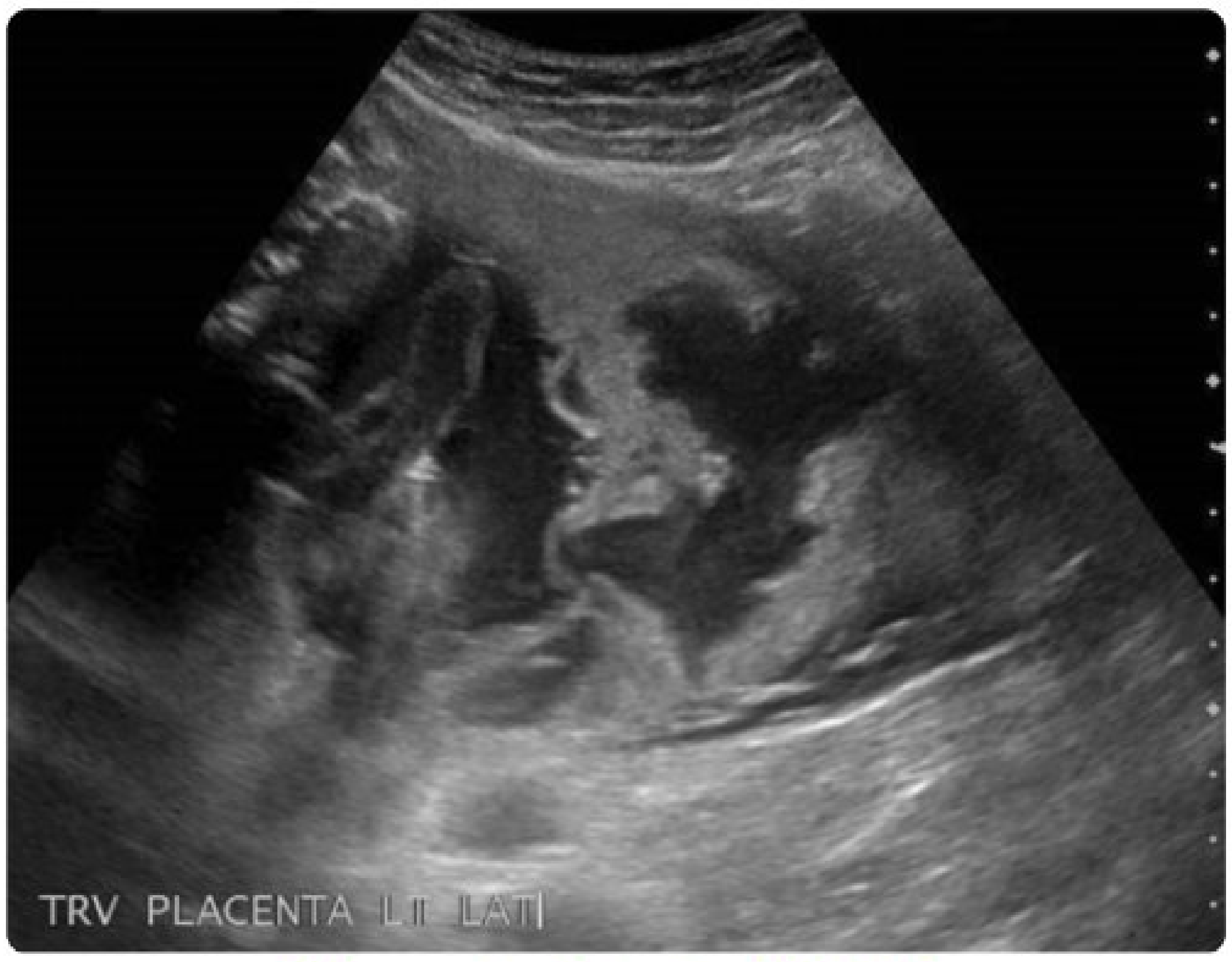
* **RISK FACTOR** :-

(MC) Risk factor : Placenta previa (in pregnancy)

2nd (MC) Risk factor : H/o Caesarean section

Others :
- H/o Uterine Curettage
- H/o Uterine surgery like Myomectomy, Hysterectomy

- Presentation : REFRACTORY PPH
- Diagnos :- IOC → USG (TVS)



Placenta Acreta (M/C)

USG Findings :-

1. Subplacental Sonolucent area which represents Decidua basalis is ABSENT
2. Heterogenous appearance of placenta
3. Placental lakes are present

GOLD STANDARD INVESTIGATION: MRI

Management: Elective C.S + Hysterectomy
↓
CLASSICAL C.S

- upper Uterine C.S because Placenta accreta is very common in placenta previa →

in placenta previa, placenta is attached to lower part of uterus
↓
If incision is given on lower part of uterus leads to ↑↑ Bleeding.

*If Hysterectomy Refused
↓
leave placenta
(remove as much as possible)

whatever part cannot be removed
↓
undergoes AUTOLYSIS

Give METHOTREXATE
↓
- to prevent Gestational Trophoblastic Neoplasia

HORMONES produced by Placenta :

1. **PROGESTERONE :** In early weeks of pregnancy → corpus luteum of pregnancy is the main source
↓
8 wks or b/w 7-9 wks of pregnancy

- This function is taken over by Placenta k/a LUTEAL PHASE SHIFT

- ≥ 8wks - Main source : Placenta
- Life span of Corpus luteum : 10-12 wks
- Precursor of Progesterone is Maternal LDL and Cholesterol
- Rate of production of Progesterone is 250mg/day (In TWINS - ≥600mg/day)

- 2 Reactions in Pregnancy :

DECIDUAL REACTION

- gland becomes hypersecretory
  +
  Stroma becomes edematous
  Rich in Glycogen

ARIA STELLA Reaction

- Gland becomes hypersecretory + Changes in Nuclei
  ↓
- seen in IntraUterine Pregnancy
- also seen in ECTOPIC and MOLAR Pregnancy.

* PROGESTERONE Relaxes smooth muscles during pregnancy.

2. ESTROGEN :

* placenta cannot synthesize Estrogen using precursors from mother as it lacks enzyme : 17 α Hydroxylase
↓
17 OH Progesterone (C21 Steroid) → ANDROSTENEDIONE (C19 Steroid)
↓
Needed for ESTROGEN Synthesis

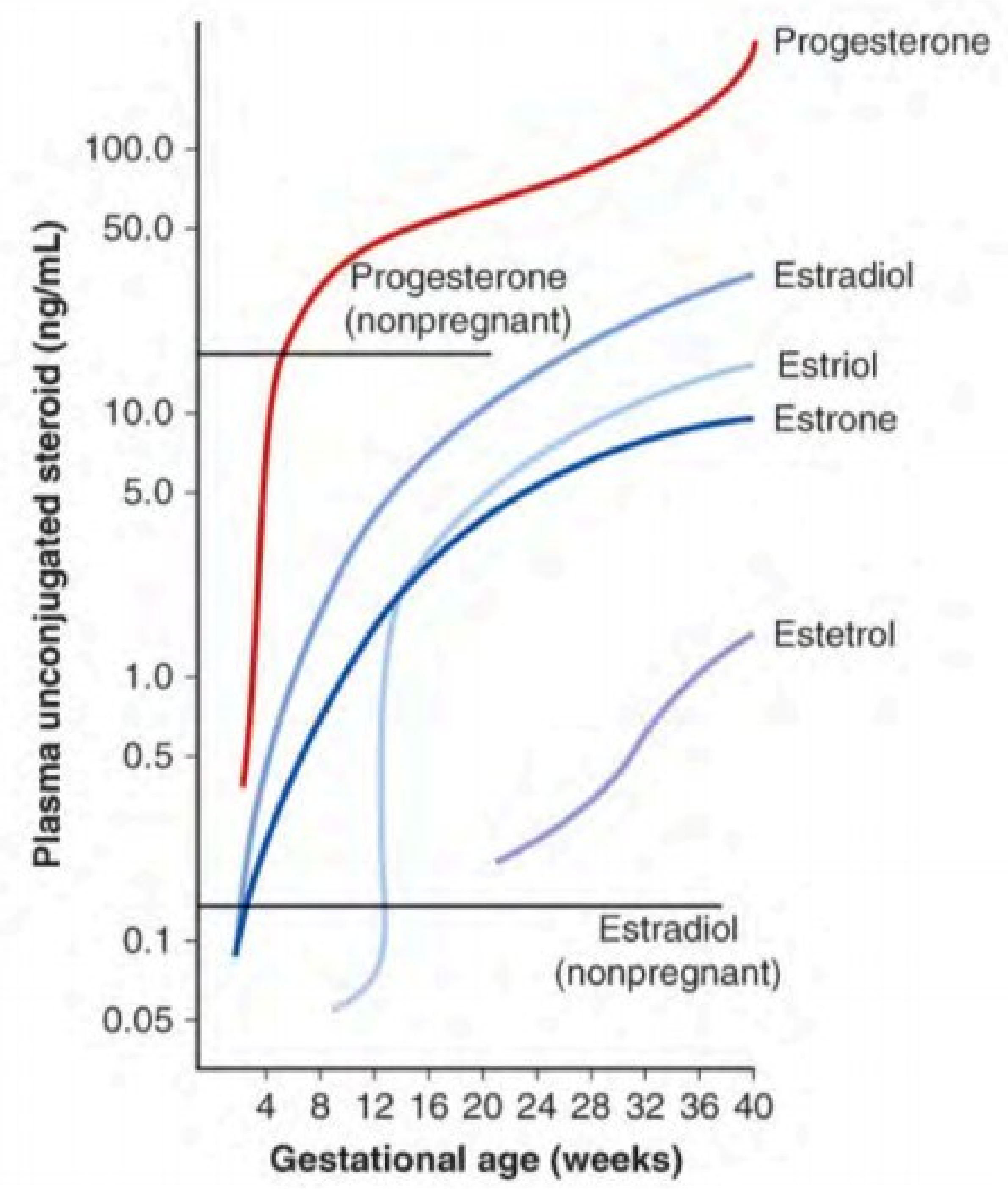
Fetal Adrenal Gland
↓ Fetal ACTH
DHEAS-S (C19)
↓
placenta will use it to form ESTROGEN
↓
Role : at term it tells uterus to contract

Enzymes :

- SULPHATASE and
- AROMATASE

SCENARIO : Suppose if pregnant female in whom, levels of Estrogen are less
↓
Estrogen will not be able to Signal the uterus to contract
↓ leads to
POST TERM PREGNANCY

- (MC) Estrogen during pregnancy } E_2 (Estradiol)
- Most specific Estrogen during pregnancy } E_3 (ESTRIOL)



↑ Estrogen	↓ Estrogen
1. Erythroblastosis Fetalis/ Rh incompatibility ⇓ ↑ placental tissue ⇓ ↑ Estrogen	1. Adrenal gland of Fetus is absent (OR) Hypoplastic ⇓ seen in Anencephaly 2. Placental sulphatase deficiency Placental Aromatase deficiency 3. Fetal demise 4. Down syndrome → overall Production of C19 is ↓ in Fetus

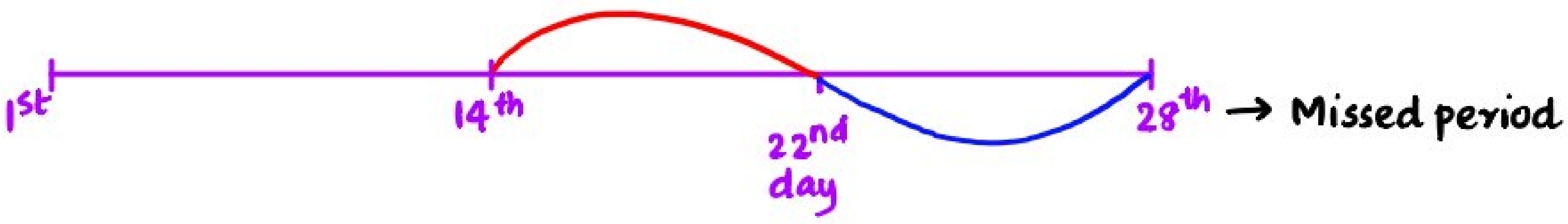
3. HCG :

Q. Main site of hormone production is SYNCTIOTROPHOBLAST

- It is a glycoprotein   HCG is the hormone with maximum Glycogen content
- It has got 2 subunits

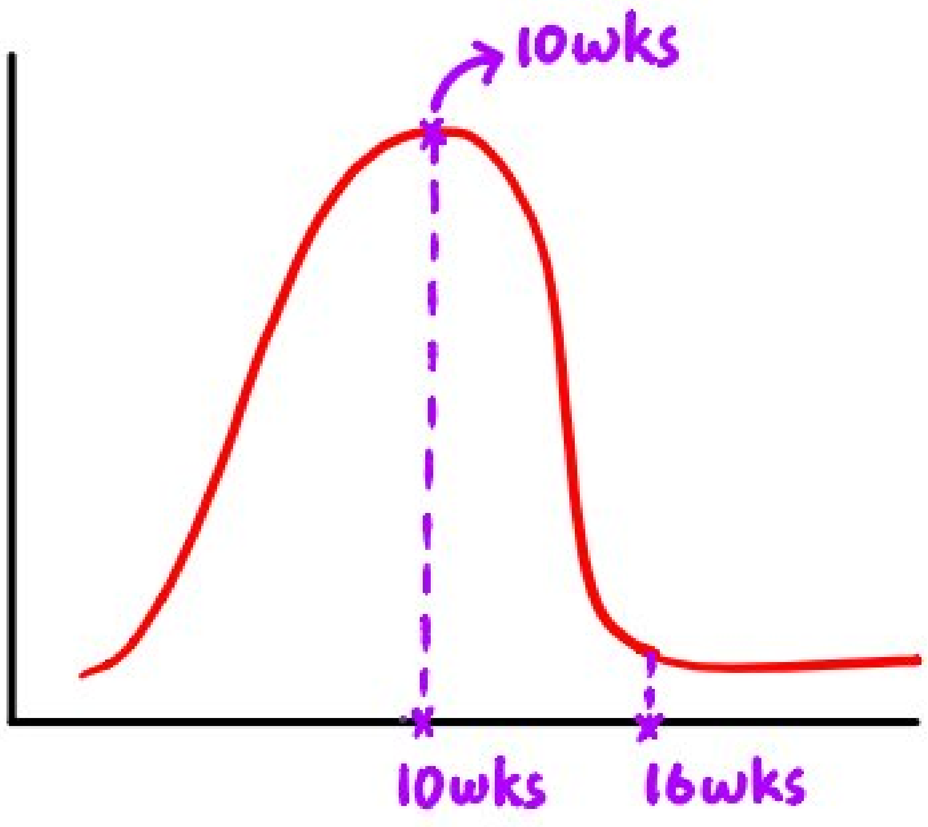
α Subunit	β subunit
• Non-specific	• Specific
• Similar to LH, FSH, TSH	• Gene → Chromosome 19
• Gene → Chromosome 6	• Measured in Pregnancy
• Not measured	

- HCG anatomically similar to LH, FSH, TSH. Functionally also similar to LH.
- HCG can be detected in blood as early as 8 days after fertilization/ Day 22 of cycle/ 5-6 days before missed period



- Most sensitive test is FIA > RIA

- levels of HCG ↑ → every 48hrs in early pregnancy – Nearly double (55-65%)



maximum HCG levels attained b/w 60-80 days of pregnancy / 20th wk of pregnancy
(HCG = 50,000 IU/L to 10^5 IU/L)
↓
After this levels of HCG ↓
↓
Minimum at 16wks of pregnancy
↓
At low levels, it continues throughout pregnancy

HCG → maintains corpus luteum in pregnancy

LH → maintains corpus luteum in non-pregnant female

- HCG levels become NORMAL → After delivery → 1-2 wks

After abortion → 2-4 wks

After partial mole evacuation → 7 wks

After complete mole evacuation → 9wks

- $t_{1/2}$ = 36hrs (LH = 2 hrs)

- Function
  - helps to maintain corpus luteum of pregnancy
  - HCG is initial stimulus for LEYDIG Cells to produce testosterone
  - prevents rejection of fetus

HCG ↑	HCG ↓
Twin Pregnancy	Ectopic pregnancy
Molar Pregnancy	All trisomies except DOWN
Choriocarcinoma	
DOWN Syndrome	Abortion
Erythroblastosis fetalis	

URINE PREGNANCY TEST

- Based on SANDWICH ELISA Test

- Sensitivity = 20 IU/L (∴ urine pregnancy test cannot detect pregnancy before missed period)

(will detect when HCG is 20 IU/L)

It can detect only at time of missed period or after missed period.

CRITICAL TITRE → That value of HCG at which in uterine pregnancy in 100% cases Gestational sac is visible.

For TVS = ≥ 1500 IU

For TAS = 6500 IU (> 5000 IU)

CASE SCENARIO I : Female - UPT ⊕ve → HCG = 1000 IU, TVS - NO Gest. sac seen

Early Intrauterine Pregnancy ↓

ECTOPIC Pregnancy ↓

Abortion ↓

Next step : REPEAT HCG AFTER 48hrs

↓ HCG lvl doubles

↓ HCG↑ but donot doubles

↓ HCG ↓

CASE SCENARIO 2: Female UPT ⊕ HCG → 2500 IU, TVS → Gestational sac not visible

↓

Case of ECTOPIC Pregnancy

4. HPL : Human Placental Lactogen aka Human Chorionic Somatotropin

- It is polypeptide hormone
- it is similar (structurally) to GH and Prolactin
- It is detected at 3wks of Pregnancy

↓ levels of HPL continue to Rise throughout pregnancy

- maximum at 36wks of pregnancy (marker for placental functioning)
- maximum production at term : 1g/day
- $t_{1/2}$ = 20-30 mins

Function
- promotes LIPOLYSIS in Mother (Glucose is spared for fetus)

↓

↑ Fatty acid → used by mother for energy requirement

- Bring about Insulin Resistance → Levels of Glucose ↑
- **INSULIN RESISTANCE :** mainly by HPL
- others - Estrogen, Progesterone, cortisol

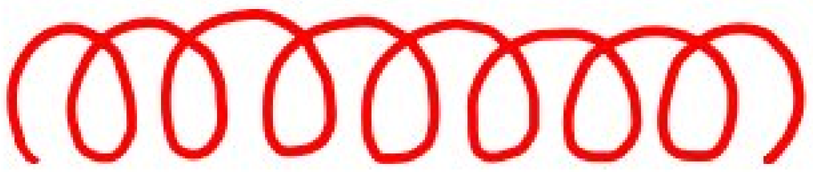
FETAL MEMBRANES

1. **CHORION** – • formed 8 days after fertilization
   • by CYTOTROPHOBLAST • Outer layer
2. **AMNION** – • derived from ectoderm • formed by 10 days after fertilization
   • innermost membrane • Avascular • Maximum Tensile strength
3. **YOLK SAC** – site for Hematopoiesis (FIRST)
4. **ALLANTOIS** – Diverticulum from HIND gut and grows in the connecting stalk

Q. Which PG is predominant in Fetal membrane – PG E_2 → during labour if membrane is ruptured
↓
PG E_2 is released → Accelerates process of labour

UMBILICAL CORD

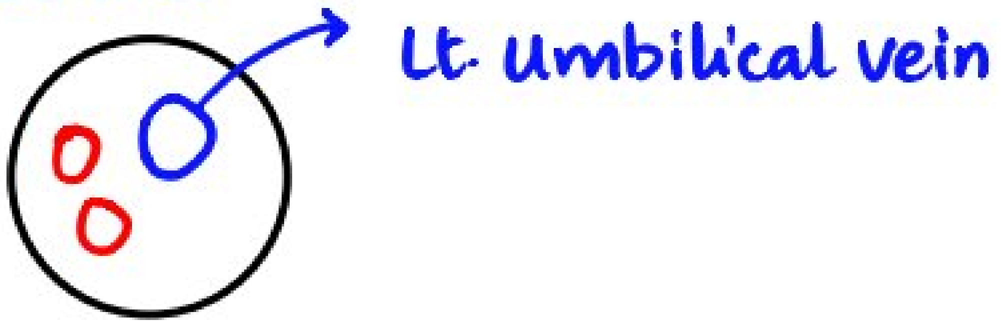
- derived from connecting stalk
- Avg. length = 55cm
- Range → 30-100 cm
- < 30cm – SHORT CORD
- pH = 7.2
- connective tissue → WHARTON'S JELLY


: FOLD OF HOBOKON

- In Early Intrauterine life = 4 vessels are present $\begin{bmatrix} \text{Rt. u.A} & \text{Rt. U.A} \\ \text{Lt. u.A} & \text{Lt. U.A} \end{bmatrix}$

  – Later on Rt. Umbilical vein disappears
  ↓
  Lt. umbilical vein is LEFT Behind

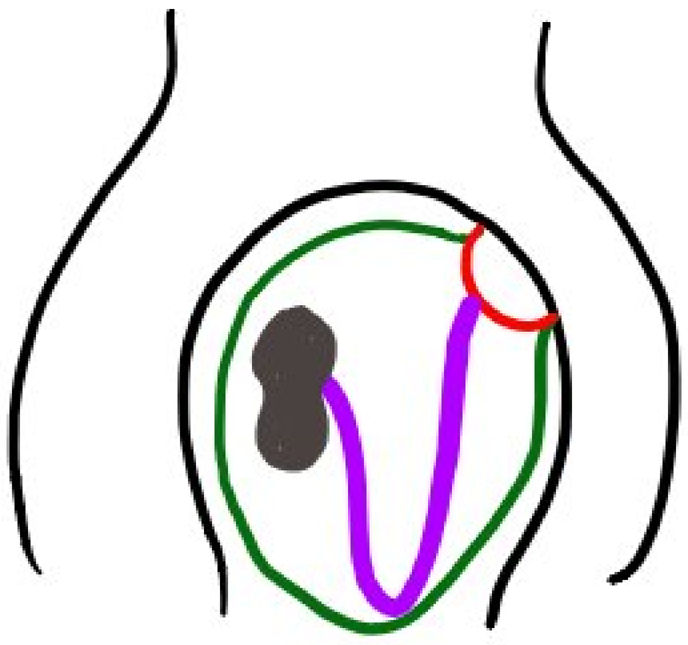
- At time of Birth = 3 vessels are present



(MC) vascular anomaly cord is SINGLE UMBILICAL ARTERY

- (MC) in Diabetes TWIN Pregnancy
- Not significant finding
- ↑ Chances of Malformations (CVS + Renal)
- Malformation + Single Umbilical Artery
  ↓
  ↑ Trisomies in Fetus

* Rarely ; **VELAMENTOUS INSERTION OF CORD**



- cord is attached to fetal membrane and then to placenta and before attaching to placenta it divides
- In this case whenever membrane will rupture

↓

cord will be exposed and gets damaged

↓

(UA and U.V) - Fetal Blood loss → ↑ Perinatal mortality

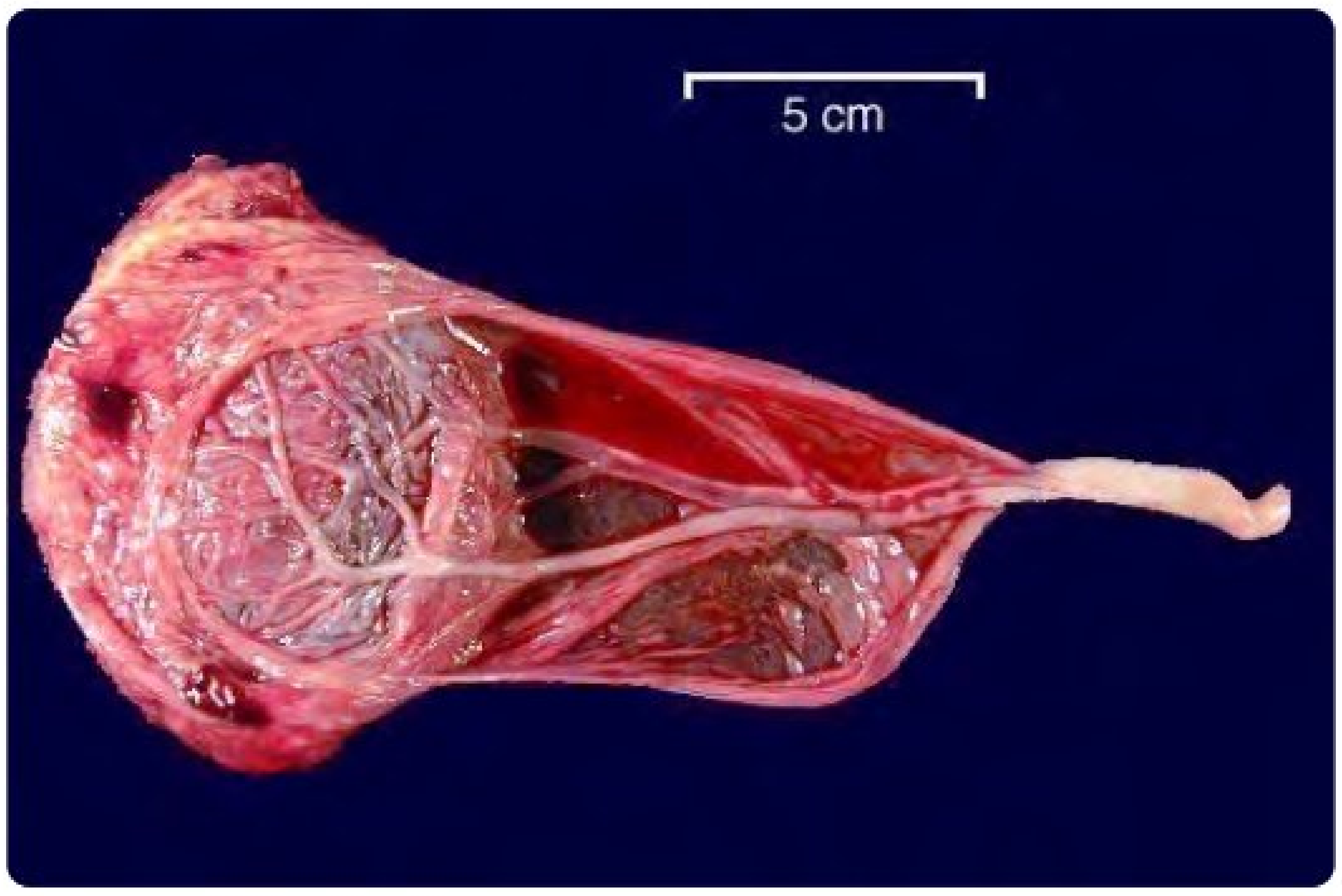
k/a **VASA PREVIA**

↓

because fetal Blood comes out through mother vagina → Confused c̄ **PLACENTA PREVIA**

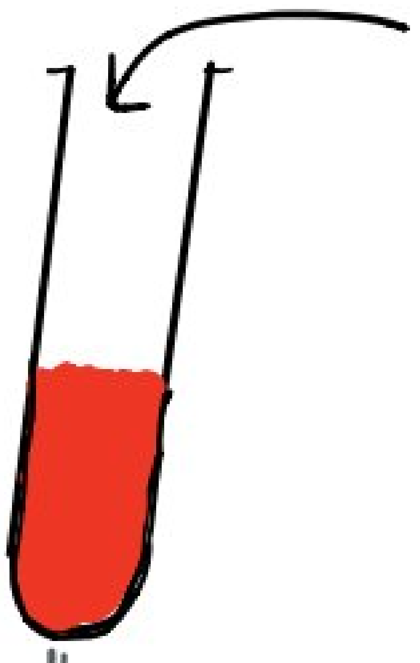
↓

SINGLE ALKALI DENATURATION TEST



- Fetal Blood
  - ↓ HbF ↓ Resistant to Acid and Alkali
  - ↳ VASA PREVIA
- Maternal Blood
  - ↓ HbA ↓ Sensitive to ACID and ALKALI
  - ↳ Placenta previa

Test :- APT test / singer Alkali denaturation test



Reagent NaOH / KOH

⇓

If colour Remains same

↓

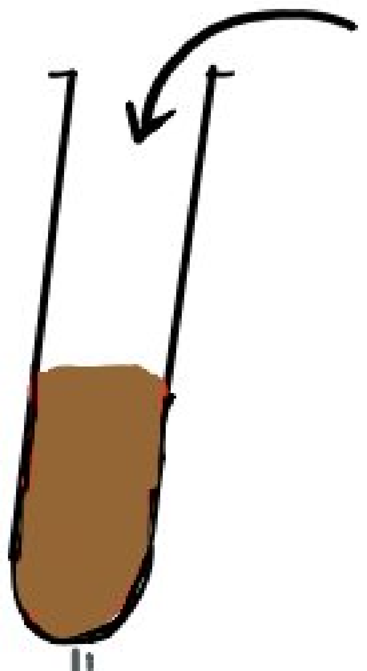
blood Resistant to Akali

↓

HbF ⊕

↓

VASA PREVIA



Reagent to NaOH / KOH

⇓

If it become Brown colour

↓

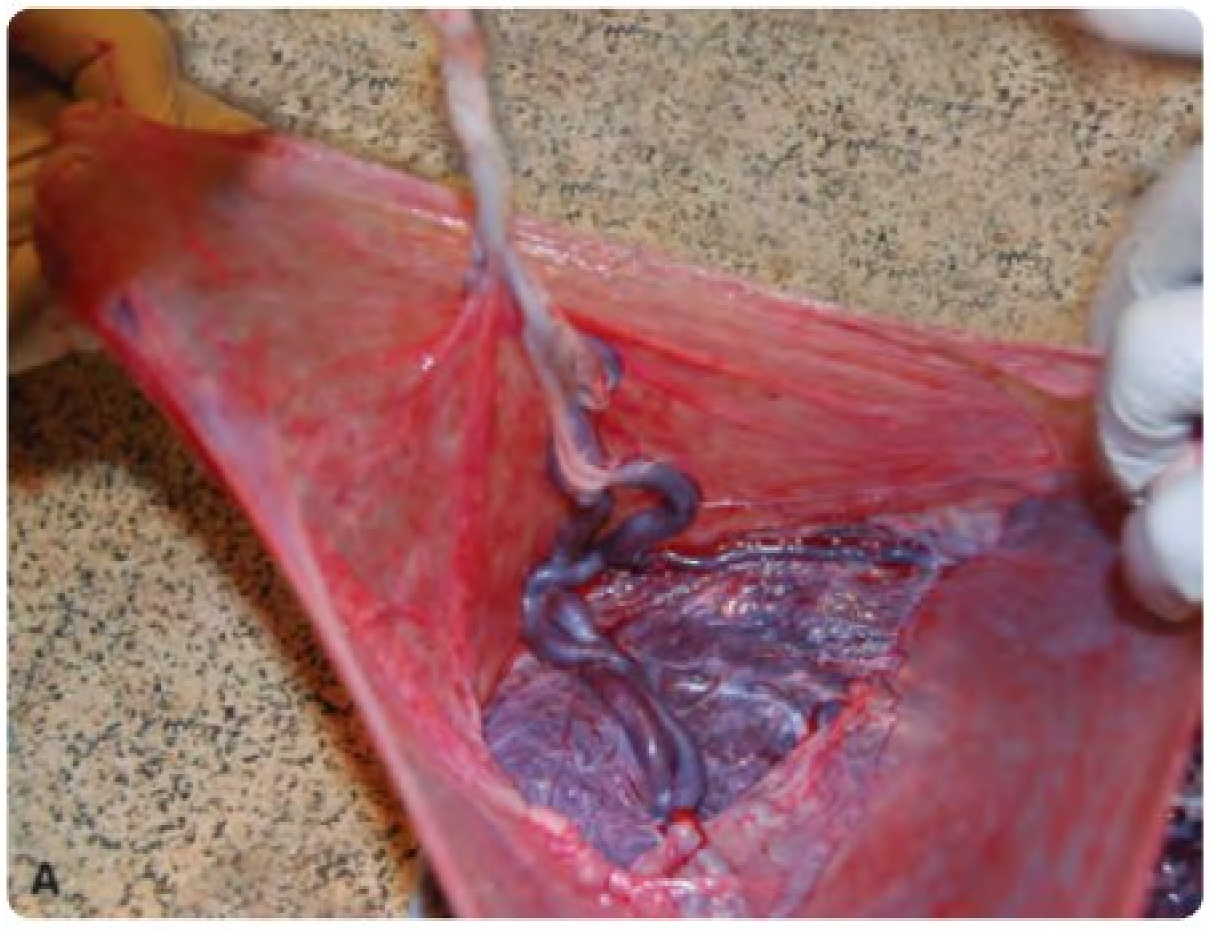
Hemolysis Ocured

↓

Hb A ⊕

↓

PLACENTA PREVIA



Velmentous cord insertion

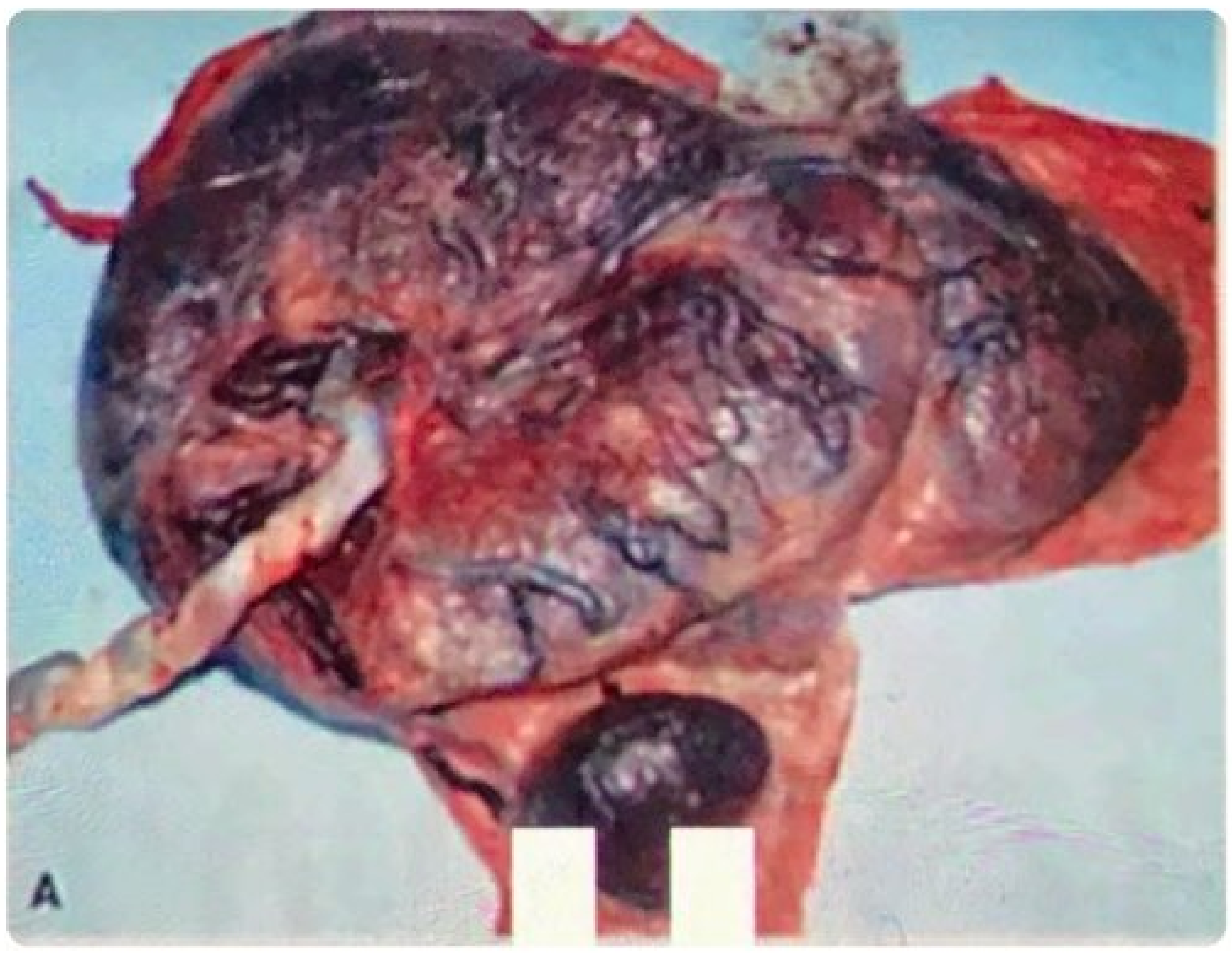
→ **Diagnosis** : In ANTENATAL Period → DOPPLER USG

→ **Management** - Cesarean Section

→ **Prognosis** - ↑ Perinatal mortality

QUESTIONS

Q. Identify the condition :



a. Placenta Bilobata

b. Placenta succenturiata

c. Circumvallete placenta

d. Circummarginate placenta

Q. Median Umbilical Ligament is a remnant of

a. umbilical artery b. umbilical vein c. Urachus d. Gubernaculum

Q. The 2 umbilical arteries near the insertion of cord to placenta are connected by

a. Funis anastomosis b. Hyrtle anastomosis c. Hobner anastomosis

d. Hanis anastomosis

Q. Folds of Hoboken are found in

a. The amnion b. The placenta c. Uterus d. umbilical cord e. Ductus venosus

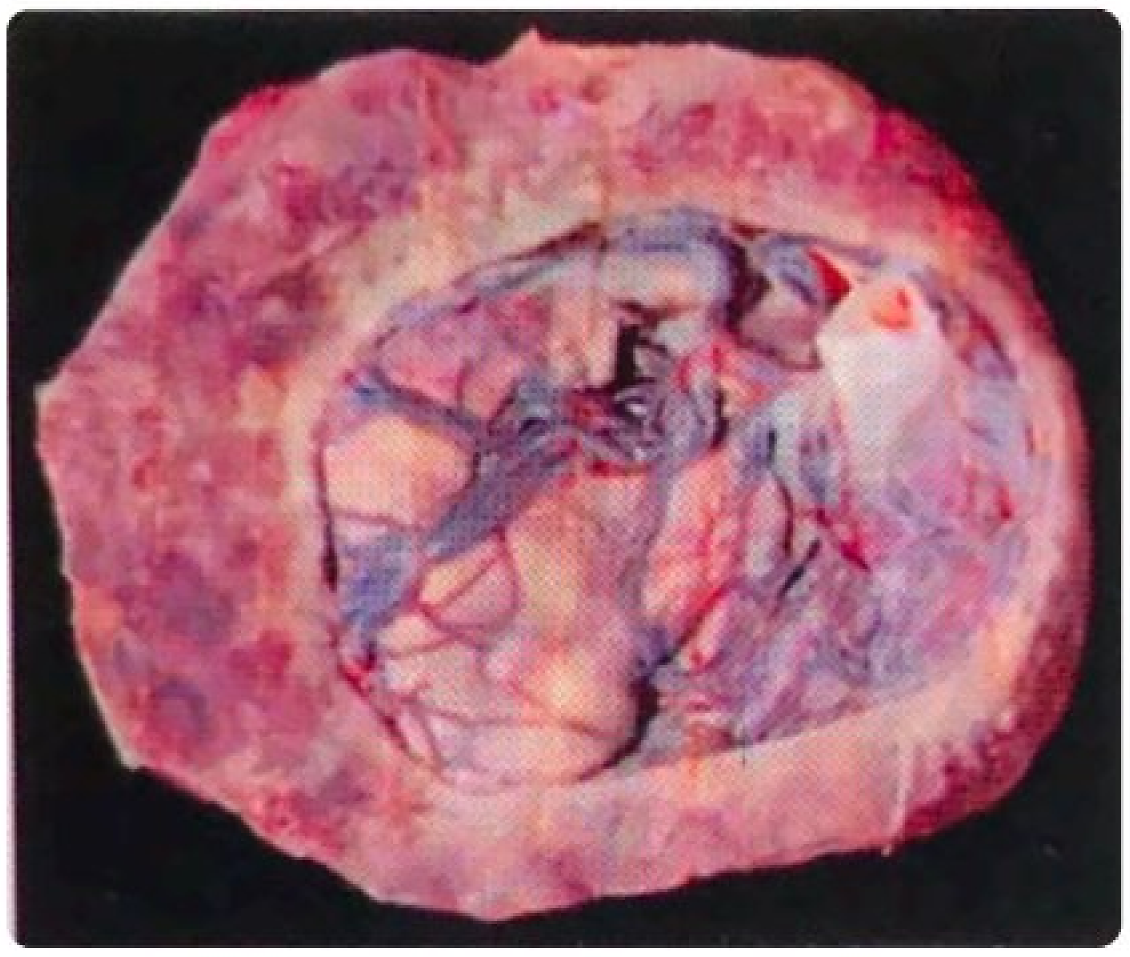
Q. Fetal Blood loss is Abnormal cord insertion is seen in

a. vasa previa b. Battledore placenta c. Decidua basalis d. Succenturiate placenta

Q. Human placenta is best described as

a. Discoidal b. Hemochorial c. Deciduate d. All of the above

Q. Identify the type of placenta shown in figure :



a. Placenta Succenturiata b. Fenestrated placenta

b. Circumvallete placenta d. Circummarginate placenta

Q. Uteroplacental Blood flow at term is :

a. 300 - 500ml/min b. 500-700ml/min

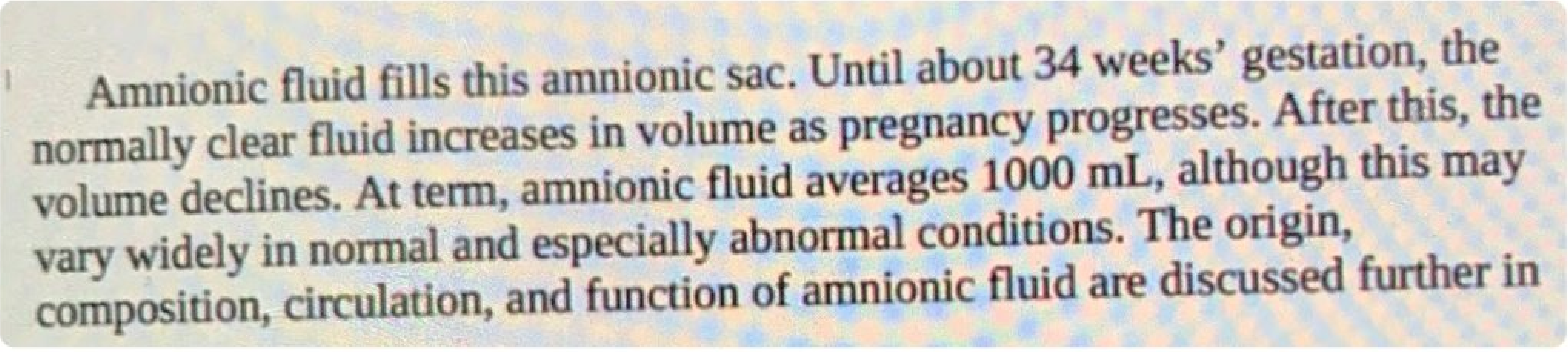
c. 700 - 900ml/min d. 900 - 1100ml/min

Q. Weight of fetus and placenta are equal at

a. 10wks b. 15wks c. 17wks d. 24wks

AMNIOTIC FLUID AND IT'S DISORDERS

- Specific gravity – 1.008 to 1.010
- Osmolality - 250 mOsm/L
- Rate of Turn over - 500ml/hr
- pH – 7.2 to 7.4 (alkaline)
- Volume of Amniotic fluid - Maximum at 34wks of pregnancy (32-34wks)



Amnionic fluid fills this amnionic sac. Until about 34 weeks' gestation, the normally clear fluid increases in volume as pregnancy progresses. After this, the volume declines. At term, amnionic fluid averages 1000 mL, although this may vary widely in normal and especially abnormal conditions. The origin, composition, circulation, and function of amnionic fluid are discussed further in

- As pregnancy advances after 40wks → Volume of AF rapidly decreases

Major Contributors of AF :

Period Gestation	Major Contributor
Early wks (1st TM)	Maternal plasma
12-20wks	Fetal skin
≥ 20wks	Fetal urine

Q. Overall - Fetal urine

Q. Urine production begins in fetus at 12wks

Q. Keratinization of fetal skin occurs at 22-25wks pregnancy

• Normally AF is colourless and at term = It is STRAW COLOUR

COLOUR	Seen in
Green colour (meconium)	• Fetal stress • Breech presentation / Transverse lie Q. • LISTERIA Infection
Golden colour (Bilirubin)	• Rh incompatability
Tobacco juice	• Intrauterine death
Saffron colour (Yellowish green)	• Post dated Pregnancy
Dark colour	• Abruptio placenta

* Besides producing amniotic fluid, fetus also swallows amniotic fluid, that is how amniotic fluid is kept in balance

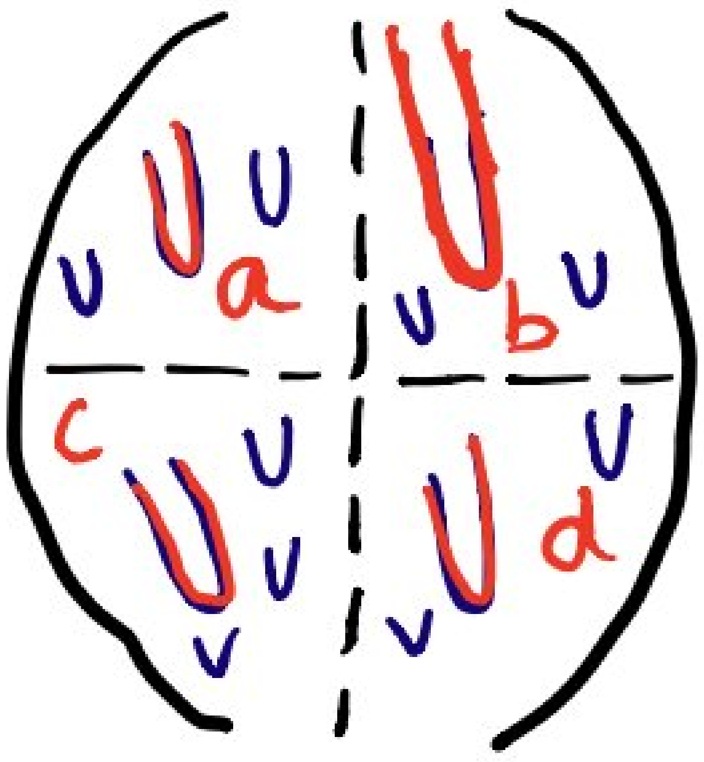
* Composition → 99% water

* Function → Performs a number of functions except NUTRITIVE FUNCTION

Measurement of Amniotic fluid

USG : measure the largest vertical AF pocket in each quadrant

↓



add a+b+c+d → AMNIOTIC FLUID INDEX (AFI)

↓

AFI = 5-24 cm (NORMAL)

- If < 5cm - OLIGOHYDRAMINOS
- If > 25cm - POLYHYDRAMINOS

- BEST / MOST SENSITIVE MARKER

↓

measure the single largest vertical pocket (Pocket 'b' in diagram)

↓

Normally = 2-8cms

< 2cm
↓
OLIGOHYDRAMINOS

≥ 8cms
↓
POLYHYDRAMINOS

* Volume of AF < 200ml - OLIGOHYDRAMINOS

≥ 2 Litres - POLYHYDRAMINOS

OLIGOHYDRAMINOS	POLYHYDRAMINOS
MC cause of MILD → Idiopathic ↳ of Severe → Congenital Malformation ↓ Renal anomalies	MC cause of Mild → Idiopathic ↳ of Severe ↴ GI Malformation > NTD GI Malformation ↓ • Any defect which not allows fetus to swallow eg: Cleft lip NTD ↓ • d/t CSF leaks into AF • a/w Swallowing defect.
Other causes: D = Drugs - Indomethacin; ACE inhibitors I = IUGR (d/t Brain sparing Effect) L = Leaking following Amniocentesis M = Maternal High BP/PIH ↓ Volume of Blood going to fetus ↓ ↓ Fetal Blood flow ↓ ↓ GFR ↓ P = PROM P = Post term pregnancy A = • Amnion nodosum • Chromosome Abnormalities TRIPLOIDY and TRISOMY 13 R = Renal anomalies in fetus	Other causes: * Whenever urine production is ↑ by fetus: ↴ 1. TWIN / MULTIFETAL Pregnancy 2. Maternal diabetes ↓ Fetal Hyperglycemia ↳ Polyuria 3. High Fetal Cardiac output conditions like; i) Fetal anemia; • Rh incompatibility • Parvoviru infection • Hemolysis ii) α Thalassemia iii) Fetal Barter Syndrome * Whenever fetal swallowing is defective; • Oesophageal atresia • Duodenal atresia

NEET 2020

Q. A 22yr old Primi c̄ sure of dates, visits ANC OPD c̄ 20wks POG, on further obstetric examination uterine height reveals 16wks size. Obstetric USG shows reduced liquor. What is the probable cause?

a. Renal agenesis b. Fetal anemia

c. Barter's synd. d. Liddle Synd.

- Intestinal Obstruction
- Cleft lip, Cleft palate

TRISOMY 21

TRISOMY 18 = IUGR + Polyhydraminos

* when CSF leaks into amniotic fluid
↓
NTD

* causes : which should mugged up
i) Chorangioma of placenta

ii) TORCH infection

EFFECT OF OLIGOHYDRAMINOS :

IF IT OCCURS EARLY IN PREGNANCY

1. Less space → fetal parts will not grow properly

(MC) → Pulmonary Hypoplasia

2. AMNIOTIC BAND SEQUENCE

- If Amniotic fluid is less → Amnion wraps around some body structure like a band leading to devascularization of that part

(MC) : LIMB Amputation > Craniofacial defect
↓
By this time complete organogenesis
↓
But problem remains same i.e less space
↓
CORD gets compressed (has umbilical artery)
and vein
↓
Blood supply to fetus decreases
↓
Fetal distress
↓
Fetus Pass meconium in AF
↓
1. MECONIUM ASPIRATION SYNDROME

EFFECT OF POLYHYDRAMINOS

1. If amniotic fluid is INCREASED
↓
Membranes gets stretched

Pre-Term Labour PROM

2. Mother experiences Respiratory discomfort

3. Abruptio placenta
↳
↑ Amniotic fluid ↓

Rupture of membranes
↓
Entire AF comes out suddenly
↓
Size of uterus shrinks
↓
placenta detaches from uterus
↓
ABRUPTIO PLACENTA

4. CORD PROLAPSE

5. MALPRESENTATION

6. AMNIOTIC FLUID EMBOLISM

2. COMPRESSION DEFECT → CTEV

- time of delivery in Idiopathic Oligohydraminos → 37wks to 38+6 days

Management of Oligohydraminos

AMNIOFUSION → with Ⓝ Saline

Q. Amnio infusion is indicated in all except:

a. Oligohydraminos

b. Fetal distress

c. Augmentation of Labour

d. washout suspected meconium

7. ↑ chances of PPH (as tone of uterus decrease)

Management of Polyhydraminos:

Serial Amniocentesis:

↓

Indication: only if patient having a lot of Respiratory distress

* Drugs which can be used to manage polyhydraminos

↓

INDOMETHACIN

↓ (helps in Closing Ductus Arteriosis)

* should not use Indomethacin beyond 32wks of Pregnancy

↓

* because before 32wks of pregnancy chances of closure of ductus arteriosis by using Indomethacin are very less and Reversible.

* After 32wks, chances of closure of ductus arteriosus by using Indomethacin are ↑ by >50%

∴ Indomethacin is C/I beyond 32wks of pregnancy

QUESTIONS

Q. Golden colour Amniotic fluid s/i

a. Rh incompatability

b. Foetal death

c. IUGR

d. Fetal distress

Q. The major contribution of AF after 20wks of gestation:

a. Ultrafiltrate and maternal plasma

b. Fetal urine

c. Fetal lung fluid

d. Fetal skin

Q. The pH of AF is (AIIMS)

a. 6·8 to 6·9

b. 7·1 to 7·3

c. 7·4 to 7·6

d. 6·7 to 6·8

Q. All of the following are functions of AF except,

a. maintenance of fetal temp.

b. Allows for free movement of fetus

c. provides proteins to fetus

d. Shock absorber

Physiological Changes in Pregnancy

I. Hormones of Pregnancy = PROGESTERONE + ESTROGEN

1. PROGESTERONE

Source : in early pregnancy

- CORPUS LUTEUM upto 8-10wks
- HCG prevents LUTEOLYSIS and maintains the corpus luteum of Pregnancy
- Corpus luteum of Pregnancy regress completely by 12 wks

beyond 8-10wks - PLACENTA

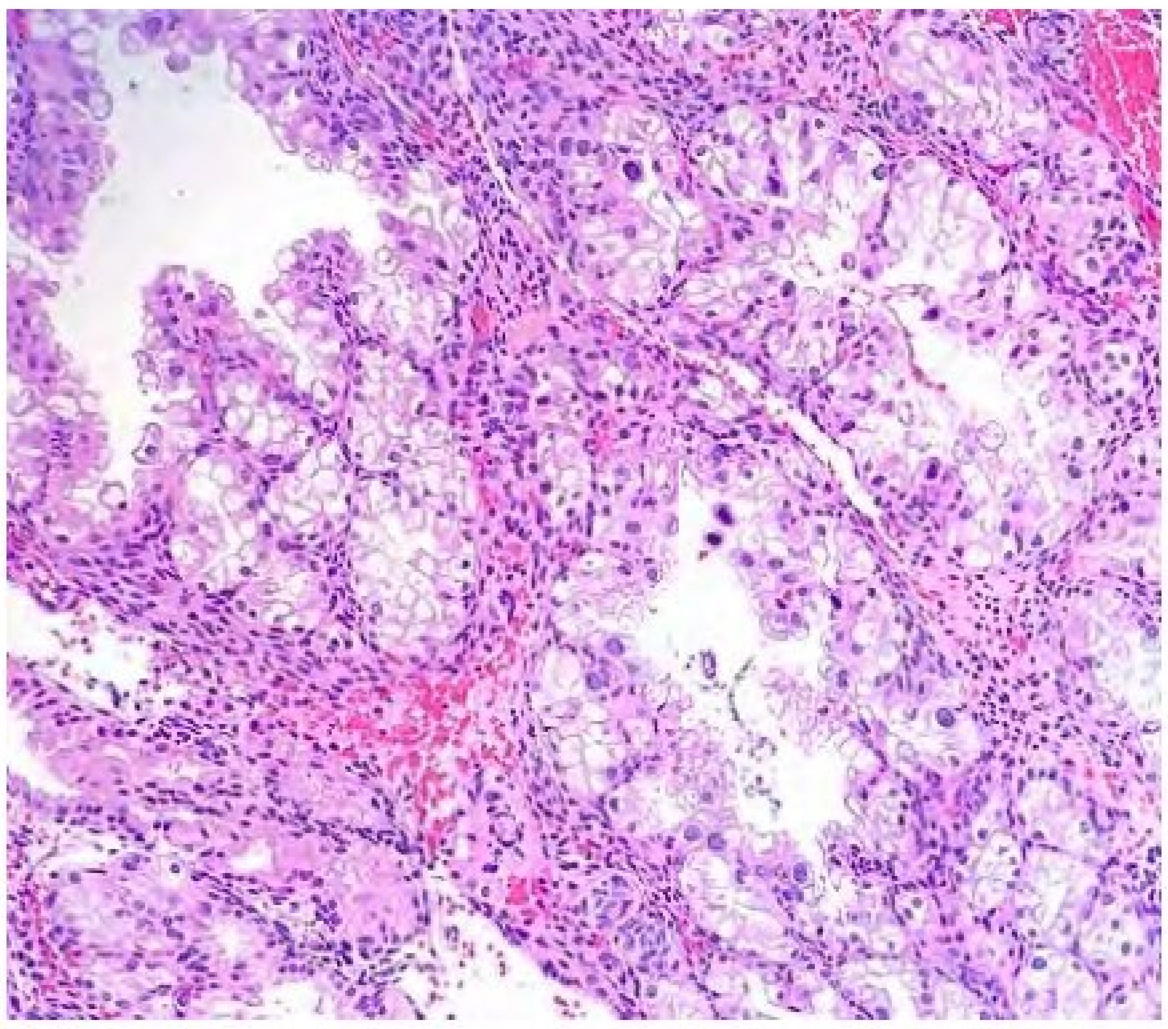
- Placenta uses Maternal LDL Cholesterol to produce Progesterone

Progesterone converts Endometrium to Decidua : DECIDUALISATION

Thickness of Endometrium at the time of implantation : 10-12 mm Ⓝ

On HPE :

- Hypersecretory change in the endometrium which looks like cancer k/a ARIAS STELLA Reaction (hormone responsible PROGESTERONE)



- Progesterone suppress Myometrial contractions and prevents Abortion maintains pregnancy

LUTEAL PHASE DEFECT : there is decreased Progesterone level

presents c̄ Repeated / Recurrent Abortions.

Rx : Progesterone Supplementation

For woman at High Risk of Pre-Term Labour (< 37 wks)

→ Give Prophylaxis Progesterone [not a tocolytic agent]

2. ESTROGEN

E_1 : ESTRONE

E_2 : ESTRADIOL

E_3 : ESTRIOL Specific to Pregnancy

Source: * Placenta cannot synthesize Estrogen on its own because it lacks 17 α- Hydroxylase

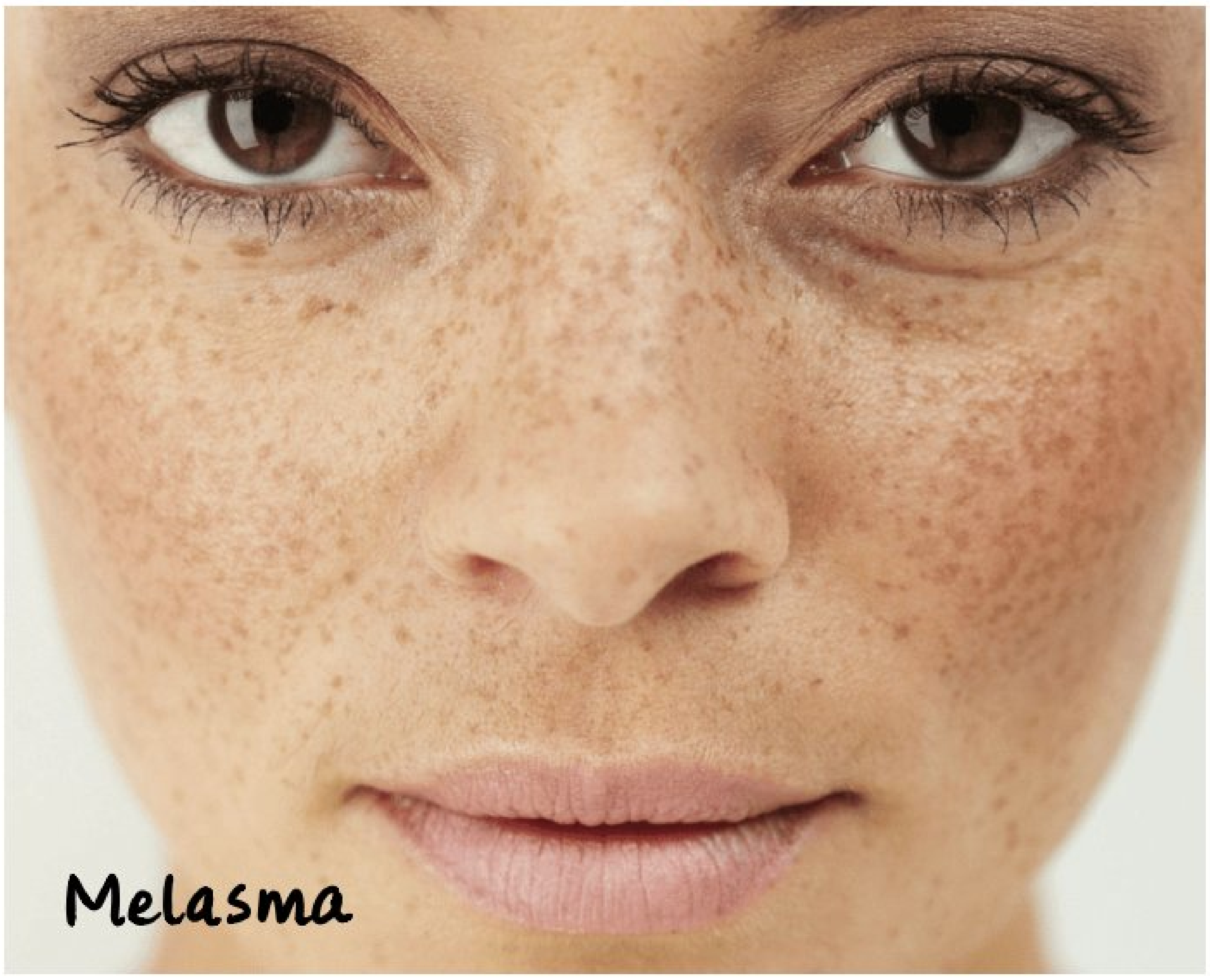
* Fetal DHEAS comes Fetal Adrenal Gland

* Fetal DHEAS reaches placenta

$$\downarrow \text{Sulfatase}$$

$$\downarrow \text{Aromatase}$$

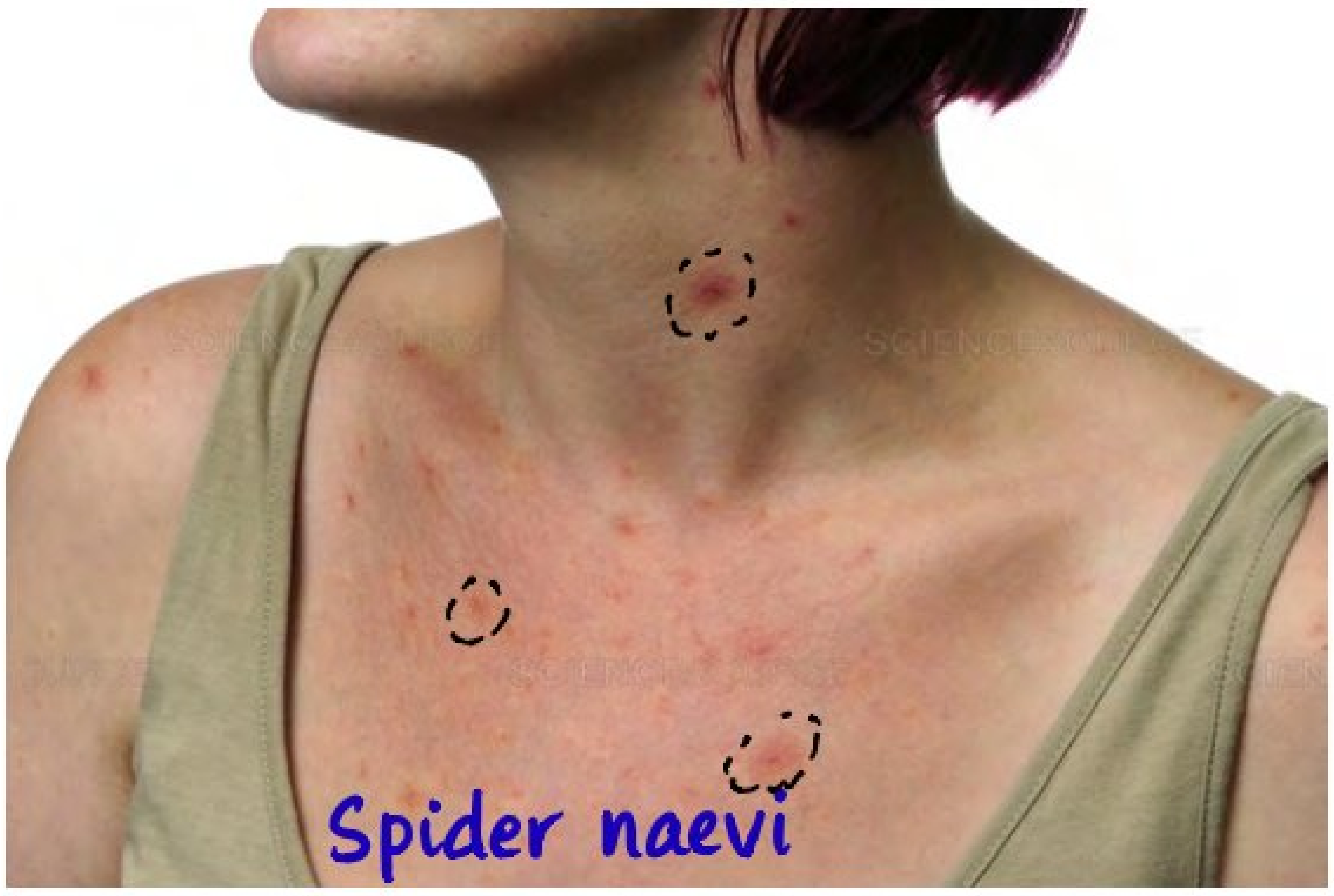
ESTROGEN [E_3]

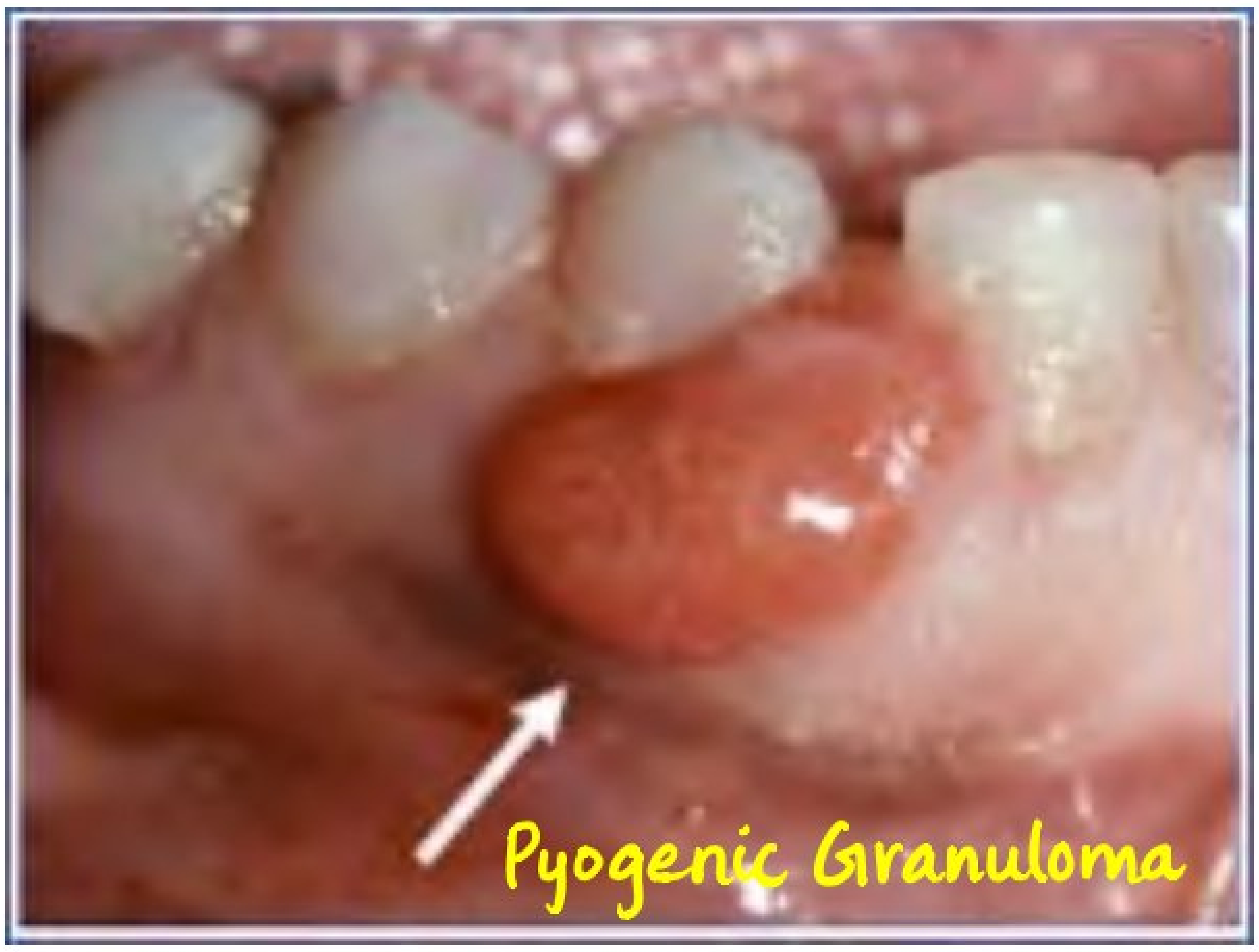


Melasma

Due to ESTROGEN following occurs

1. Melasma
2. Spider naevi





3. Pyogenic Granuloma – vascular tumor
   - * Gingiva (MC)
   - * Mucous membrane
   - * Mucocutaneous junction
4. Salt and Water Retention [6.5L]
   - there is fall in plasma osmolarity by 10 mOsm
5. Increased TBG [Thyroid Binding Globulin] in Pregnancy.

UTERUS

- muscular organ - smooth muscle

1. Endometrium - lined by Single layer of columnar cells
2. Myometrium
3. Perimetrium - serosa

- Pregnant Uterus - undergoes both HYPERTROPHY (predominant) and HYPERPLASIA

* after pregnancy uterus undergoes INVOLUTION

* for complete involution takes 6wks (i.e to 80g)

- Weight of Uterus
  - non-pregnant uterus
    - primi 50-70g
    - Multi 80g
  - Term uterus 1100g

- Volume of uterine cavity
  - Non-Pregnant Uterus - 10ml
  - Pregnant Term Uterus - 5000ml

PREGNANT UTERUS

Lower Uterine Segment [LUS]

- formed by ISTHMUS of Uterus
  - Ⓝ Uterus - 0.5cm
  - Term Uterus - 5cm

- at term - 70% of LUS made by Isthmus

30% of LUS made by Cervix. Cervix will be taken up EFFACEMENT

- described in %

- shortening of cervix
- softening of cervix

Cervix is made up of connective tissue Collagen (10-15% Smooth muscle)

- decrease in Collagen

- increase in water and Hyaluronic acid

In LSCS
- incision is given on LUS
- here Serosa is tightly attached to uterus but at uterovesical fold of peritoneum loosely attached.

PREGNANT CERVIX

- Endocervix is lined by Tall Columnar Epithelium
- Ectocervix - part we see on Per Speculum examination
  - lined by Stratified Squamous Epithelium
  - vagina pH - Acidic
- In Pregnancy, there is eversion of Columnar epithelium onto squamous epithelium → k/a **ECTROPION** it bleeds on touch - No biopsy to be done

II. Hematological

- WBC count ↑ (upto 15,000), in immediate Post Partum (upto 25,000)
- DLC - Neutrophilia
- ESR
- CRP

} both increased

- platelet count (Avg. Platelet count ↓ but ≤ in Ⓝ Range)
- Clotting factors : all clotting factors ↑ except 11 and 13 ↓ Hypercoagulable state
- Hb levels : Plasma volume ↑ by 40%

Red Cell mass ↑ by 20%

} **HEMODILUTION**

compensatory changes

1) ↑ in Cardiac Output

2) ↓ in Viscosity

ODC curve shows Ⓡ shift to ensure good oxygen supply to tissue (↑ 2,3-DPG)

- C.O ↑ by 40% , tissue O_2 demand ↑ by 20%
- Arterio Venous O_2 gradient will decrease
- Fetal ODC curve shows Ⓛ shift : higher affinity for O_2

if Hb < 11g %
Hematocrit < 33% } **ANEMIA IN PREGNANCY**

if Hb < 7g % **SEVERE ANEMIA**

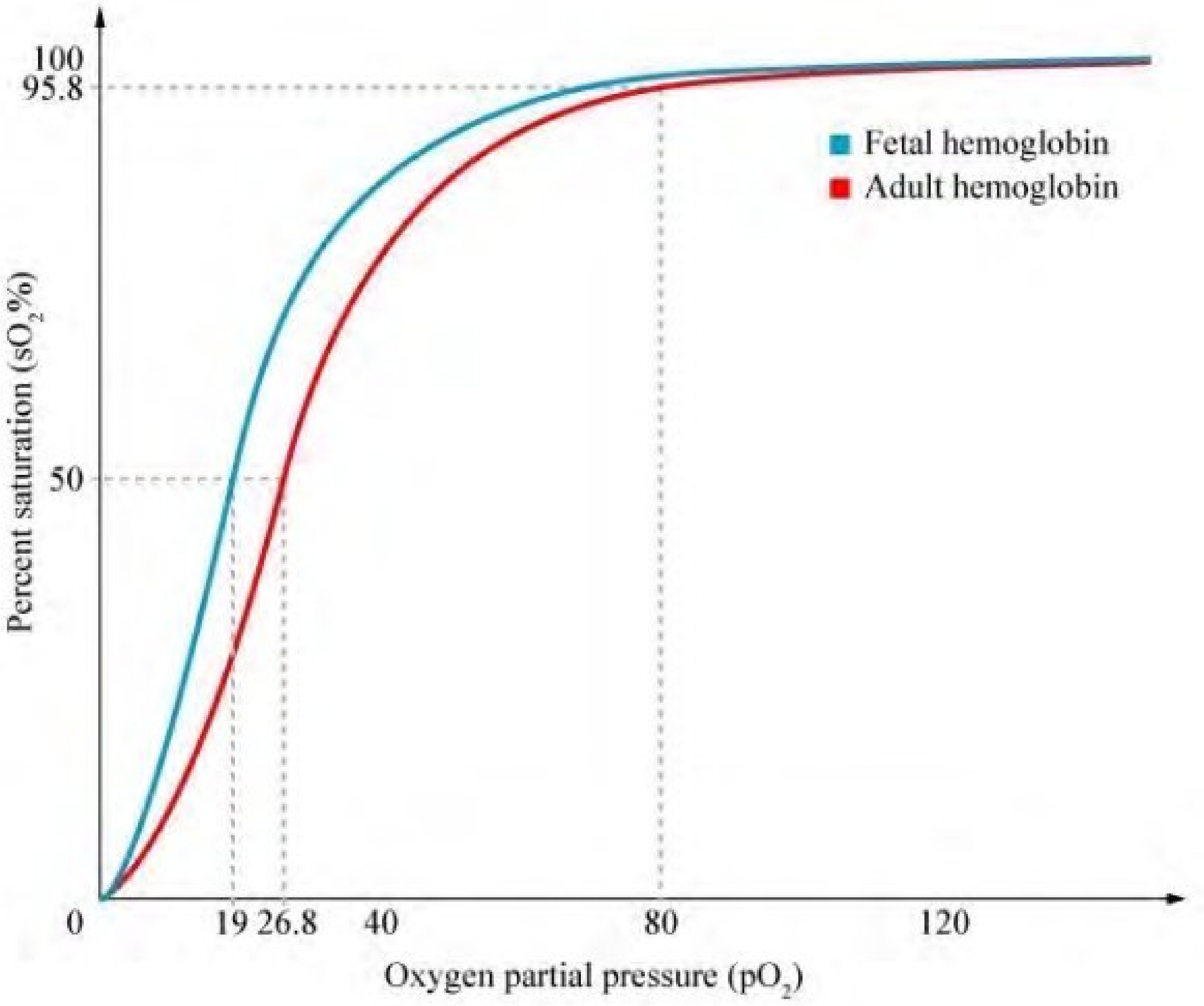
if Hb < 4g % VERY SEVERE ANEMIA

Ⓜ︎Ⓒ cause of Anemia – **Iron Deficiency**

Total Iron in Pregnancy - 1000mg

(300mg - Fetus and Placenta)

BEST MARKER for **IDA** in Pregnancy - SERUM FERRITIN



Govt. Of India

IFA Tab contains
Iron - 60mg of Elemental Fe
Folic acid - 500mg

- start giving IFA Tab at 12wks and then continue throughout pregnancy

i.e 6months + 6months Post Partum

- 1 Tab/day for prophylaxis

Q. 24wks POG ≤ Hb 6g% : Rx 2 IFA tabs/day

BLOOD TRANSFUSION done if

1. Pregnancy > 34 wks and Hb < 7g %
2. Pregnancy < 34 wks and Hb < 5g %
3. Acute Blood loss and Hb < 6g%
4. Any Hb c̄ signs and symptoms of CHF

PARENTERAL IRON given if

1. Malabsorption Syndromes
2. Non-compliant patient, if severe G.I side effect

III. CVS changes (Physiological)

- * Red Blood Cell - ↑ 20%
- * O_2 demand - ↑ 20%
- * Cardiac Output - ↑ 40%
- * Plasma volume - ↑ 40%

Cardiac Output

- * starts Increasing as early as 5wks
- * at 32wk - ↑↑
- * at Term - ↓
- * during 2nd Stage Labour - ↑↑↑
- * immediate Post-Partum Period - ↑↑↑↑ (maximum C.O)

AUSCULTATORY FINDINGS

- HR - ↑ by 20%
- all Heart Sound - LOUD
- wide Split S_1
- Systolic murmur → upto Grade 2
- S_3 heard
- Apex beat - shifts to 4th ICS (heart moves up and anteriorly to left)

On CXR

1. Apparent cardiomegaly
2. Straightening of Ⓛ Heart Border

On ECG

1. Left Axis Deviation by 15° s/i Pregnancy
2. Extrasystole

BLOOD PRESSURE

- fall in DBP and SBP (fall in DBP >> SBP)
- systemic vascular resistance (SVR) - ↓
- Afterload - ↓
- Preload - ↑ d/t expansion of Blood volume
- all Vasopressors (Renin, Angiotensin, Nor-Epi, Aldosterone) - ↑

even then there is ↓ in DBP and SBP d/t Estrogen + Progesterone

Estrogen - resistance against vasopressors

Progesterone - vascular wall relaxation

SUPINE HYPOTENSION SYNDROME

- when pregnant women lie on supine position gravid uterus compress IVC thereby ↓ in venous return and cardiac output.
- so all pregnant women >20wks are advised to lie on Ⓛ Lateral position hence there is ↑ fetal O_2 saturation by 10%
- In fetal distress : Rx Ⓛ Lateral position
- Position in LSCS : 15° Ⓛ Tilt (Wedge under Ⓡ Hip)

MC Anesthesia used in CS → **SPINAL ANESTHESIA**

- cause Hypotension
- level of Block - T_4
- pain sensation from uterus T_{10} - L_1
- given to knock off Peritoneal Nerve Supply

PAINLESS LABOUR - EPIDURAL ANESTHESIA

- level of Block for vaginal delivery : T_{10}
- MC used drug: Bupivacaine (0.0625% → 0.125%)

not beyond 0.125% → because this also cause Motor Block

IV. Respiratory changes

- ↑ Tidal volume [this is d/t Progesterone - it increases the sensitivity of Respiratory centre to CO_2]
- ↑ Minute ventilation by 40%
- ↑ Depth of ventilation (HYPERVENTILATION)
- pCO_2 - will fall
- pO_2 - will increase

leads to **Compensated Respiratory Alkalosis** by increasing excretion of HCO_3^- } No change in pH

- diaphragm is pushed up by 4cm by increasing Transverse and AP diameter
- TLC - no change (slight fall)

* RV/ERV - ↓

* VC - no change

* Pulmonary Capillary Wedge Pressure - no change

NO CHANGE in

1. RR
2. pH
3. TLC
4. VC
5. PCWPr

V Urinary Tract changes

* Kidney - ↑ in length (by 1cm)
↑ in Renal Blood Flow (by 80%)
↑ in GFR (by 50%)

S. Creatinine
S. Uric acid
BUN
} all DECREASE

S. Creatinine
S. Uric acid
} Both INCREASE in PRE-ECLAMPSIA

Hydronephrosis / Hydroureter

* is seen d/t Progesterone → suppress ureteric peristalsis

(MC) seen on Ⓡ side > Ⓛ side
↓
because Pregnant uterus is DEXTRORATED and compress Ⓡ ureter more at pelvic brim

Urinary stasis

* asymptomatic bacteriuria - has to be treated
if not treated (in 25% pt can cause pyelonephritis)

Glycosuria can be seen because, in pregnancy Renal Threshold is increased.

VI GIT

* GERD - d/t Progesterone - it decreases tone of LES
Gastric pressure increases d/t Pregnant uterus
Gastric emptying time shows no change in pregnancy

* LIVER - ↓ in AST and ALT
↑ in ALP → because of ↑ Placental Isoform of Pregnancy
↑ in Protein production (↑ in Albumin and Globulin)
↓ Oncotic pressure → Edema

In Pregnancy - S.Prolactin ↑↑ - Pituitary gland increases by 135%
because of Prolactin producing cells

↓

- doesnot maintain pregnancy
- maximum levels during pregnancy
- after delivery PRL level falls by 50%
- its MILK PRODUCING HORMONE

MILK LET DOWN / EJECTION HORMONE - OXYTOCIN

SHEEHAN'S SYNDROME

- Pituitary gland - High vascular requirement
- So in SEVERE PPH - only ANTERIOR pituitary gland is affected

↓

all Anterior Pituitary horm. ↓

- MC Presentation - Failure to Lactate
- 2nd MC Presentation - Persistent Amenorrhoea

* if woman donot lactate → Return of Menses in 6-8wks

* can also present as ADRENAL CRISIS

can present c̄ signs and symptoms of
- FSH and LH deficiency
- Growth Hormone deficiency

Diagnosis of Pregnancy

Presumptive

- perceived by herself

1. Amenorrhoea
2. Morning Sickness
3. Breast tissue enlargement or tenderness
4. Increased frequency of urination

Probable

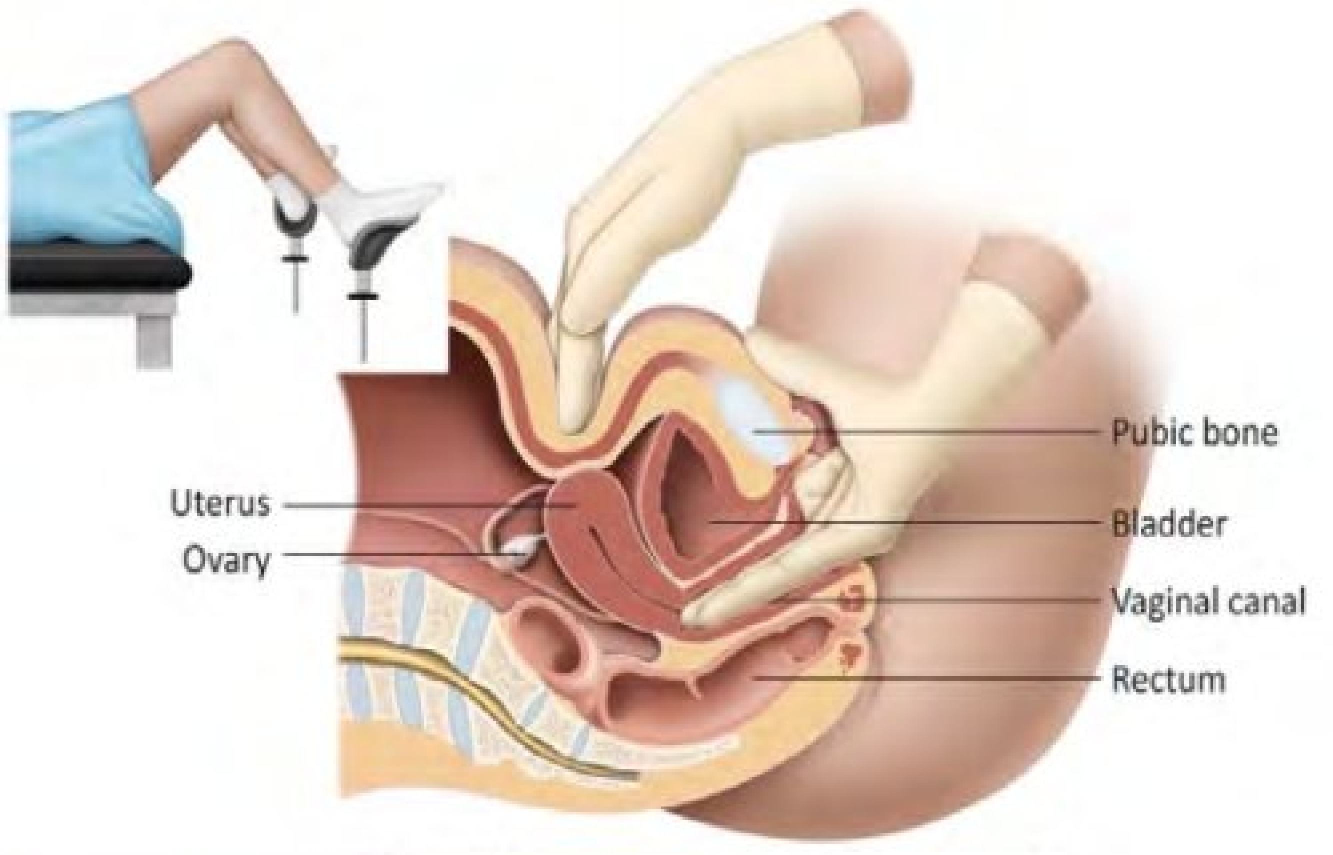
- not confirmatory
- these are signs seen by clinicians on BIMANUAL Examination.

Positive

- confirmatory

1. Fetus on USG
2. Fetal parts can be felt
3. Fetal Heart Sound can be heard

BIMANUAL Examination

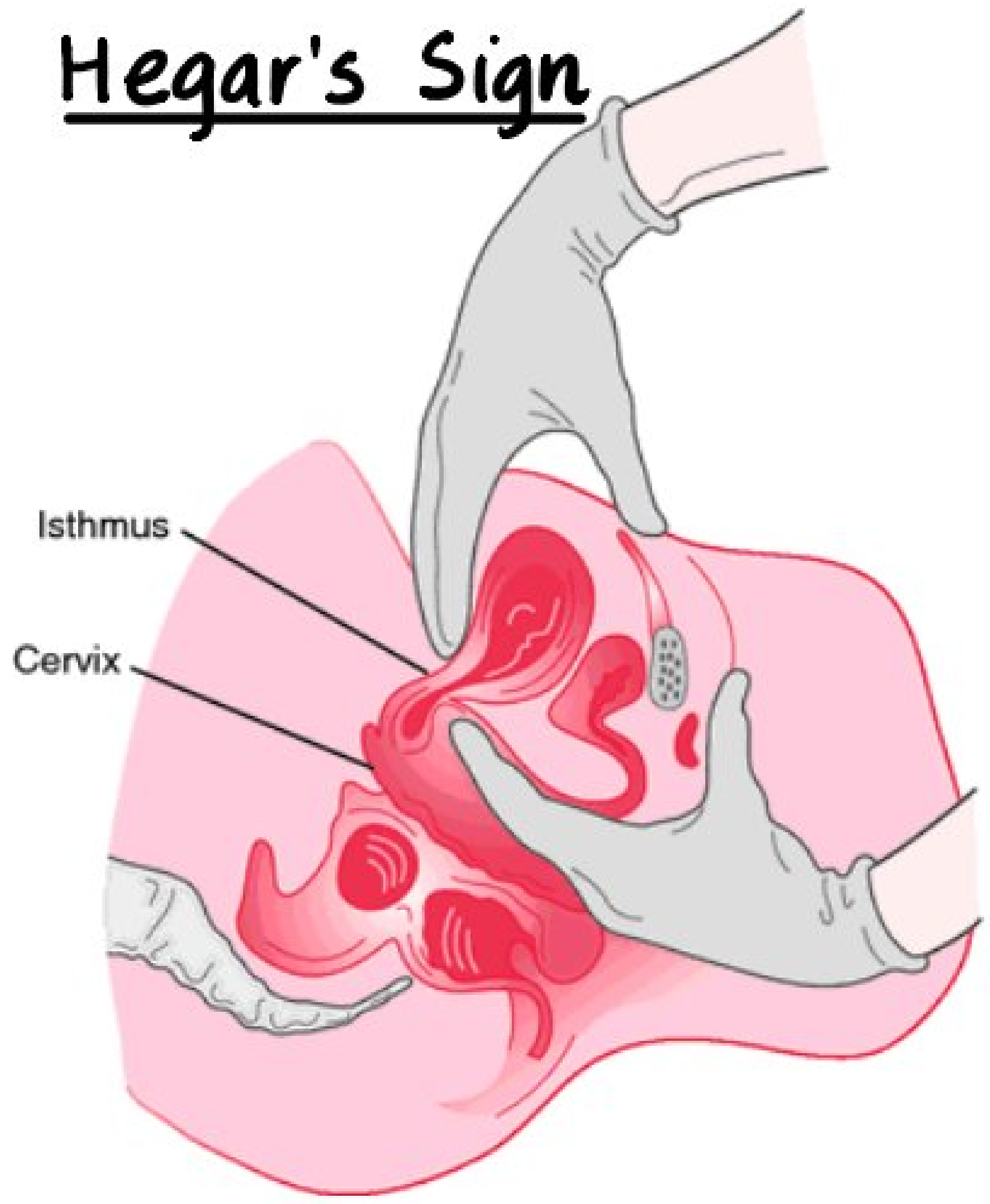


Probable Signs

HEGAR'S SIGN

- 6wks of pregnancy
- softening of uterine isthmus
- isthmus is the narrow constricted area between corpus and cervix of the uterus
- corpus and cervix are connected to each other at the level of Internal OS

Hegar's Sign



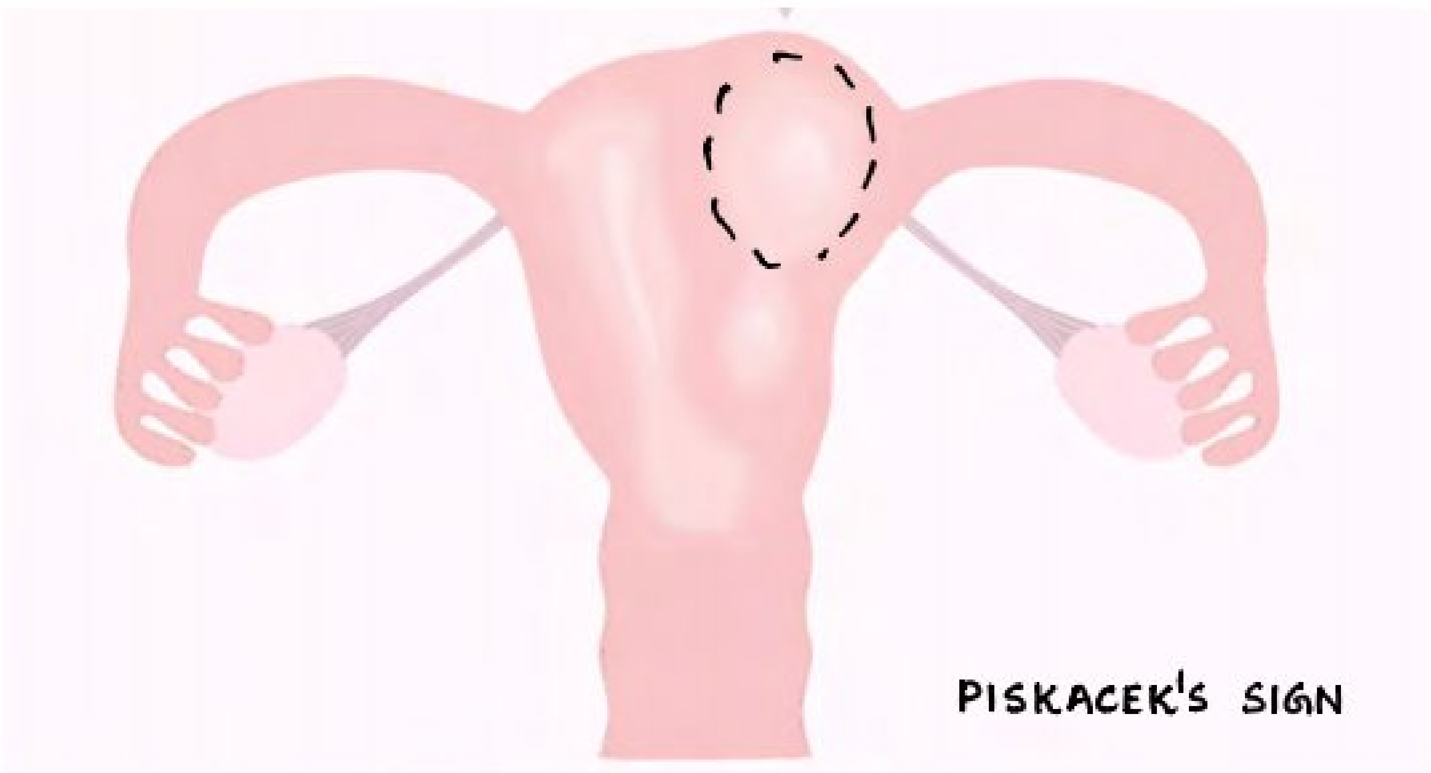
External OS is between cervix and vagina

- On Bimanual examination : Abdominal hand and vaginal hand touch each other

GOODELL'S SIGN

- 6wks of pregnancy
- softening of cervix

PISKACEK'S SIGN


PISKACEK'S SIGN

- 8wks of Pregnancy
- unequal growth of uterus, because of lateral implantation
- implantation of conceptus : always eccentric
  - ↳ Most common site : upper posterior wall

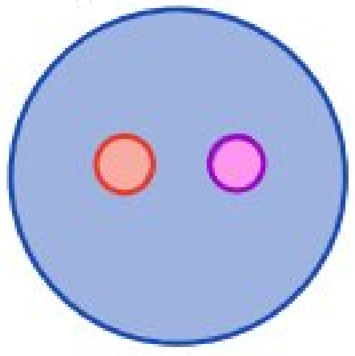
Form of conceptus implantation happens : **BLASTOCYST**

Human Embryonic Development

Fertilization of ovum : occurs in Ampulla of Fallopian Tube and remains there for 3 days

↓

Conceptus : male and female Pronucleus fuse and then divide



↓ 1st Division occurs after 30hrs after fertilization

2 celled Stage

↓

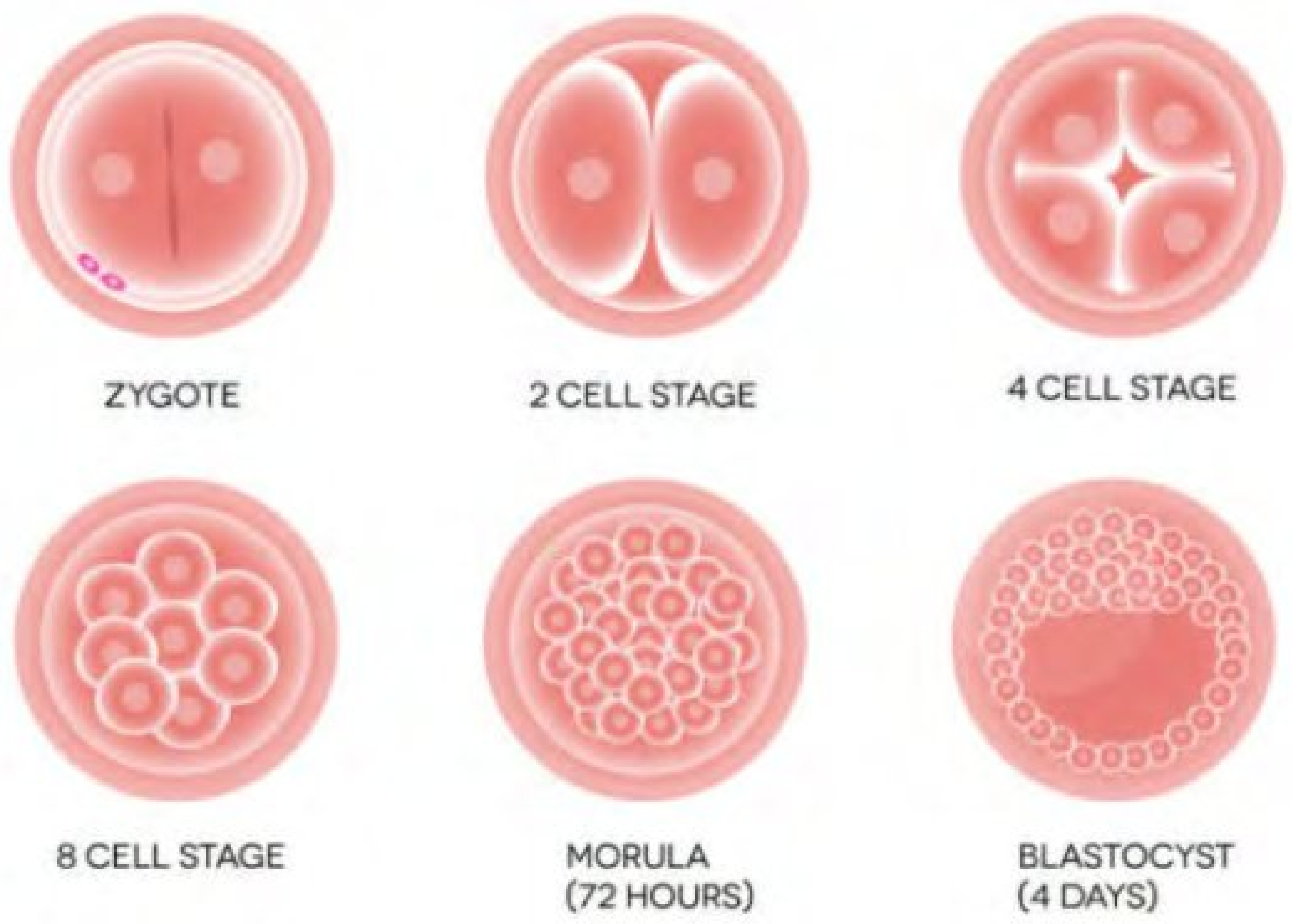
4 celled stage

↓

8 celled stage

↓

16 celled stage : **MORULA**



MORULA • form in which conceptus enters uterine cavity on 4th day after fertilization / 3 days after fertilization / Day 18 from LMP

↓

BLASTULA

↓

BLASTOCYST - formed on Day 5 after fertilization
- implantation begins on Day 6
- implantation completed by Day 7

Weeks - Period of Gestation Day - Post Fertilization

Ovulation happens on 14th day = Fertilization = 1st day of LMP

eg: morula enters the cavity on Day 4 from fertilization / Day 18 from LMP

- Implantation happens in 3 Phases
  1. Apposition - require Selectins
  2. Adhesion - require Integrins - most important
  3. Invasion - require Matrix Metallo Proteinase

Implantation Window - Day 20-24 of Menstrual cycle

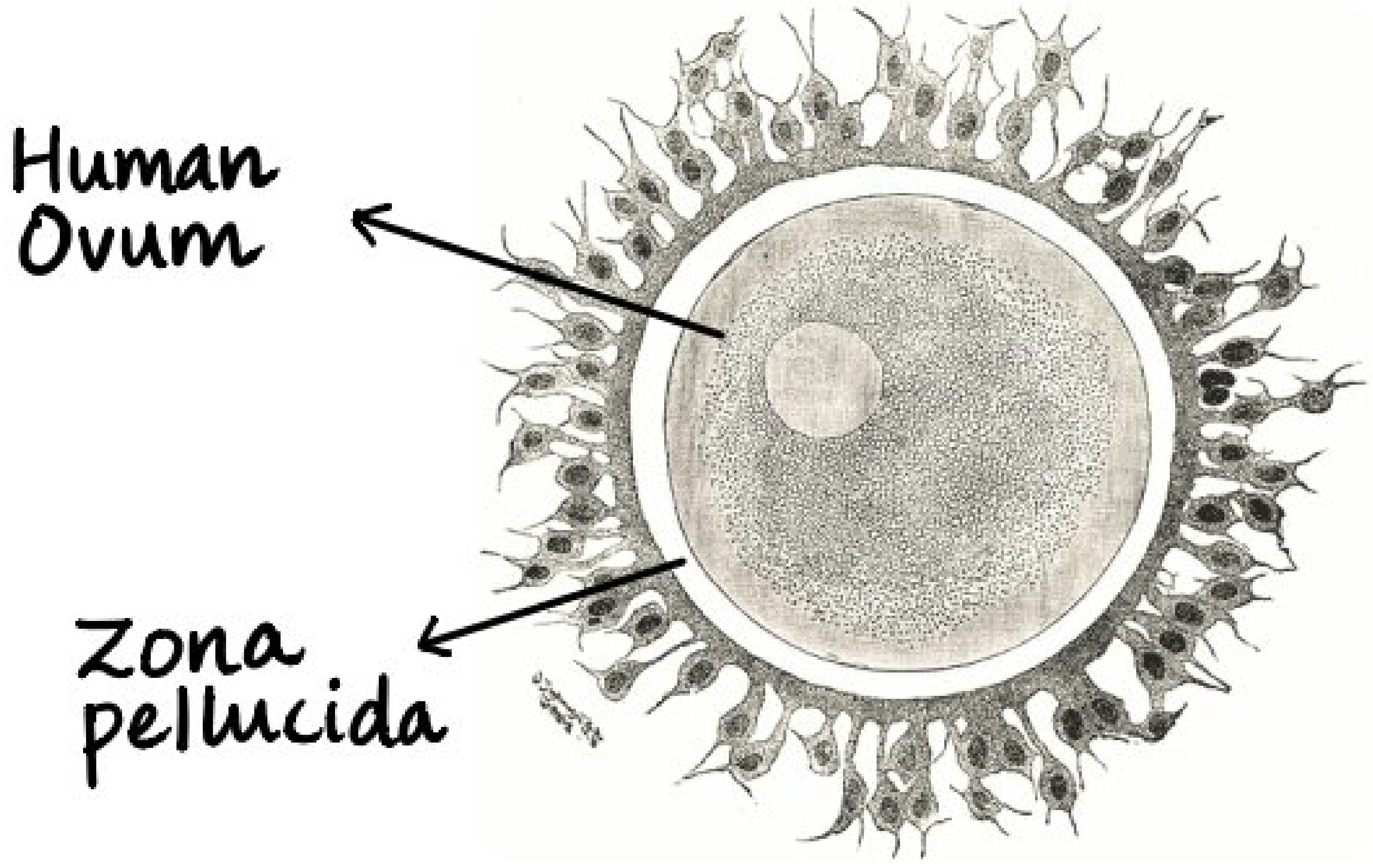
Hartman' Sign (or) Placenta Sign - small amount of bleeding at the time of implantation which is normal

ZONA PELLUCIDA

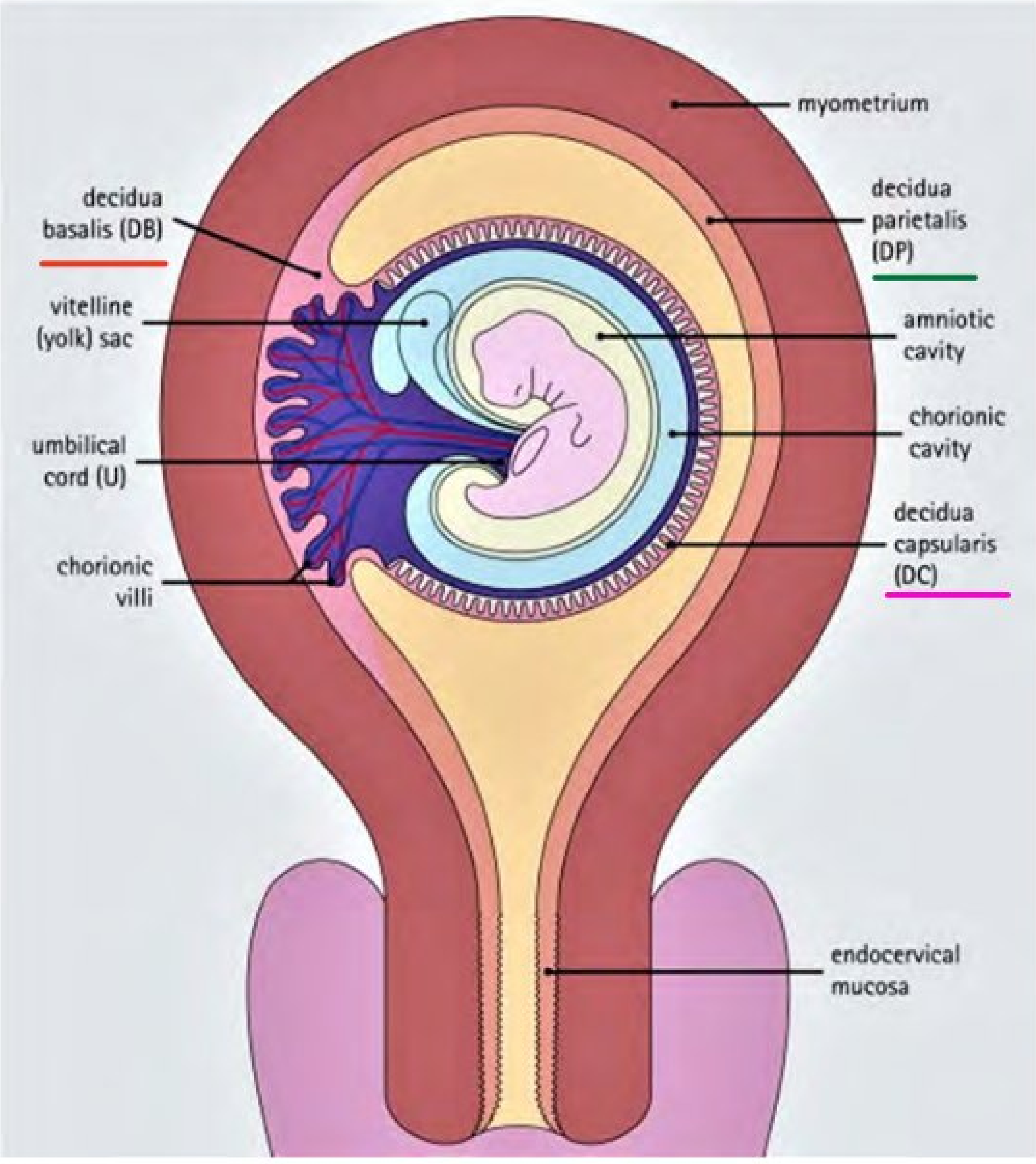
-its an acellular membrane

-made up of Glycoprotein

-Receptor for Sperm → **ZPGP-3**



- By cortical reaction it releases certain granules which prevents polyspermy
- lost on day 5 - **ZONA HATCHING**



DECIDUA

* It is endometrium of Pregnancy
* 3 layers of Decidua are
  i. Decidua basalis
  ii. Decidua capsularis
  iii. Decidua parietalis

* uterine cavity gets obliterated by fusion of Decidua capsularis and Decidua parietalis known as **DECIDUA VERA**. This occurs -16 wks of POG [14-16wks]

Fertilization of 2 Ova each by a separate sperm and by 2 different coitus

if this occurs in same menstrual cycle known as **SUPERFECUNDATION**

if this occurs in different menstrual cycle known as **SUPERFETATION**

OSIANDER's SIGN

- 8wks of Pregnancy
- it is lateral vaginal wall pulsations, because of increased vascularity to the uterus [Uterine artery]

percentage of Cardiac output to uterus at term - 10%
Blood flow to uterus at term - 750ml/min
Uteroplacental flow at term - 450 - 750ml/min

uterine artery

- is branch of Anterior division of Internal Iliac
- at level of Internal OS crosses over Ureter "Water under the Bridge"
- Uterine artery ligation is done at the Internal OS

Ovarian artery

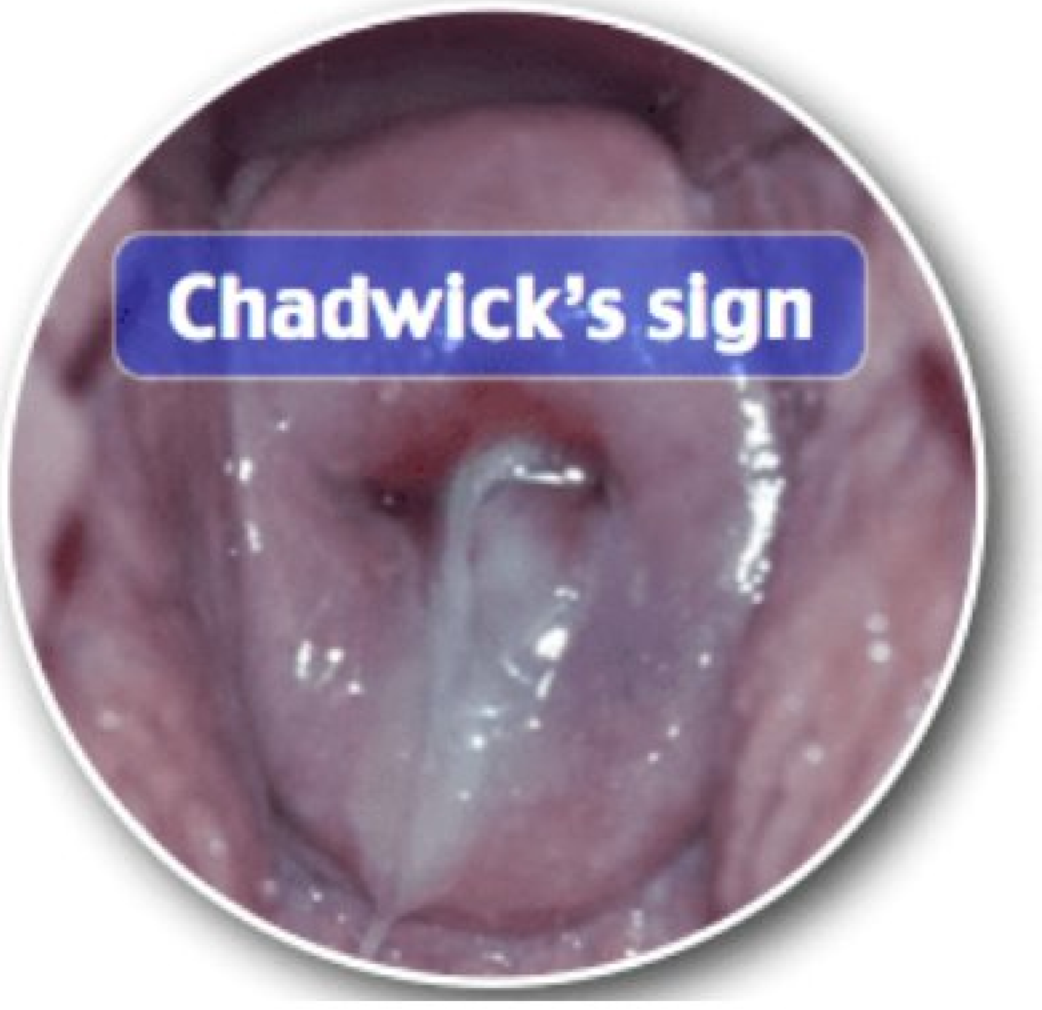
- is branch of Abdominal Aorta at level of L2
- gives blood to uterus through anastomosis dilates 3 times in Pregnancy

Ureteric injury

- MC site of ureteric injury in OBG Surgery is 2cm lateral to cervix
- 2nd MC site for ureteric injury Pelvic brim [Here ovarian artery crosses ureter]

CHADWICK'S SIGN

- bluish discolouration on vagina and vulva



PALMER'S SIGN

- regular rthymic contractions

Braxton Hicks Contractions:

donot raise intrauterine pressure hence not perceived by pt.

Pregnancy test

- all are immunologic test [Probable Sign]

1. **HCG** - Human Chorionic Gonadotropin

- its a glycoprotein hormone
- 2 Subunits - α / β

- 2 Subunits - α / β → resembles β-LH and differs only in 8 amino acids

  α: non specific and absolutely identical to FSH, LH, TSH

URINE PREGNANCY TEST

- MC done test
- less sensitive

  min amount - 20IU/L
- Qualitative test
- usually Positive 1 wk after missed period

  MC reason for negative UPT when woman is pregnant is if done too early
- Based on SANDWICH ELISA

SERUM PREGNANCY TEST

- more sentitive
- min amount - 1IU/L
- Quantitative test
- Becomes Positive much earlier i.e

  8 Days Post-ovulation or

  8 Days Post-Fertilization

Based on **DECREASING** order of sensitivity of test

1. FIA - Fluoro immuno Assay
2. RIA - Radioimmuno Assay
3. RRA
4. ELISA - Enzyme Linked Immunosorbant Assay
5. IRMA

HCG can give idea about viability:

if viable HCG in early pregnancy shows DOUBLING

How to look?

- D_1 Day1 - 1st sample
- D_3 Day3 - Repeat sample: shows a rise after 48hrs

55 - 66% rise

Abnormal Pregnancy / Non-viable pregnancy

1st Scenario D_1 - 1st sample
D_3 - 2nd sample - if fall in HCG } **ABORTION**

2nd Scenario D_1 - 1st sample
D_3 - 2nd sample - if slow rise } **ECTOPIC**
- doubling happens in 7days

β-HCG production starts as early as 8 days post fertilization

↓

Keeps Rising

↓

attains maximum value at 10wks [60-70days]

Normal value in pregnancy – 1lakh IU

↓

after 10wks it begins to fall reaches minimum value by 16wks [16-20wks] after which plateau

Conditions where HCG value is more than expected

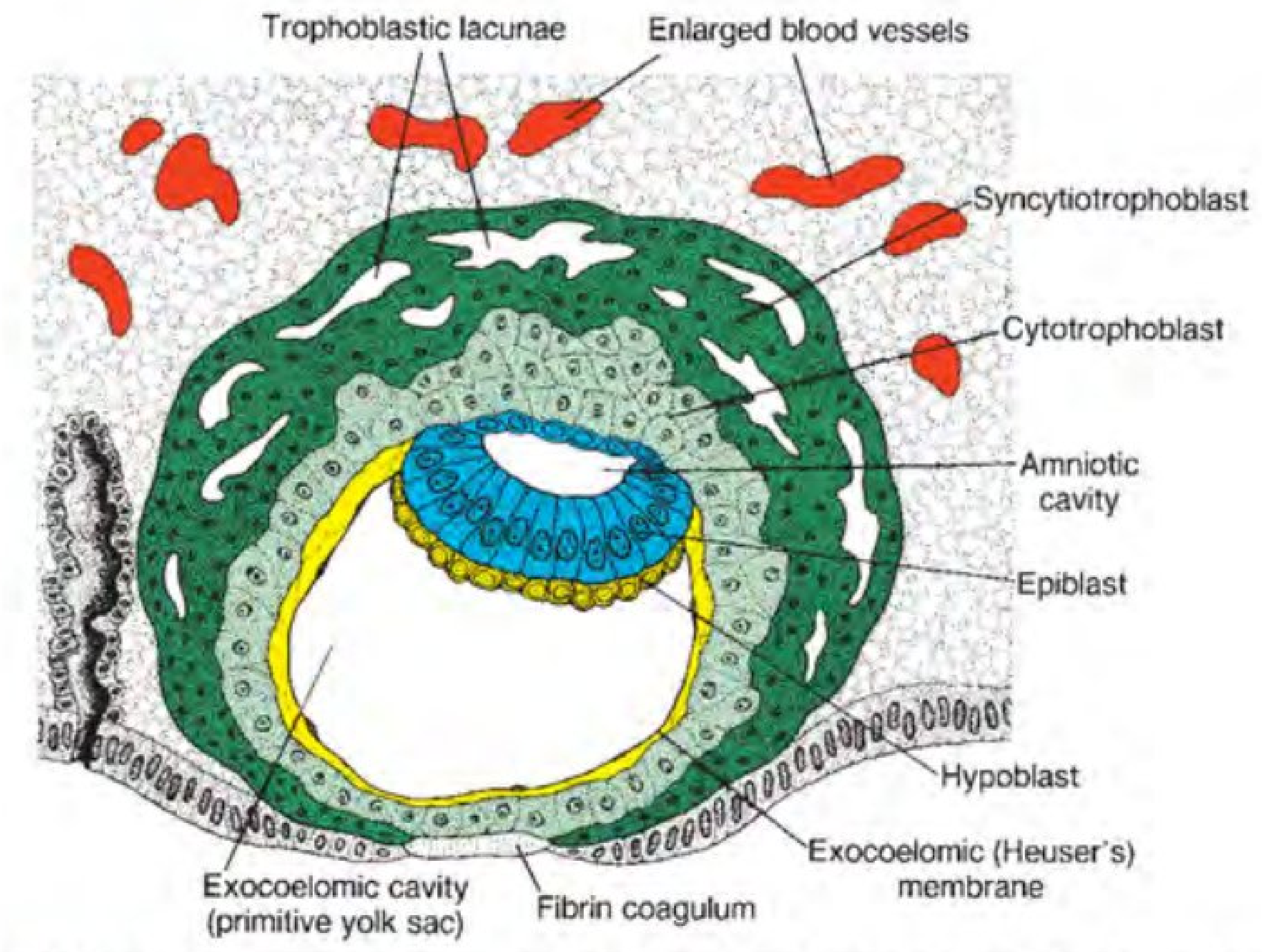
1. Gestational Trophoblastic Disease
2. Multifetal pregnancy d/t increase in Placental tissue

   HCG secreted by placenta - SYNCYTIOTROPHOBLAST

   ↓

   - from which all hormones are secreted [Steroids, proteins]
   - source of syncytiotrophoblast is cytotrophoblast
3. Erythroblastosis fetalis – placenta becomes swollen and big.
4. DOWN'S Syndrome
5. Underestimated Gestational Age
6. Hyperemesis Gravidarum

Trophoblast differentiation occurs 8days post fertilization



Conditions where HCG value is less than expected

1. Non-viable pregnancy
2. Ectopic pregnancy
3. Trisomy 18
4. Overestimated Gestational age

2. HPL Human Placental Lactogen

aka Human Chorionic Somatomammotrophin (HCS)

- can be detected by 3wks POG (Starts producing)
- produced by syncytiotrophoblast
- no α/β Subunit
- for placental functioning HPL keeps increasing as POG increases
- it is the hormone produced in maximum amount at term by placenta.
- main function - Endocrinal, responsible for Insulin resistance in pregnancy

POSITIVE / CONFIRMATION OF

1. Pregnancy
2. Location of sac
3. Viability

} done by USG

USG in Early Pregnancy : TVS > TAS → • 3-3.5MHz
- done on full bladder

TVS ↓
- more sensitive
- will pick up finding 1 week earlier than TAS
- Higher frequency ≥ 5 MHz - 7 MHz
- Done on empty bladder

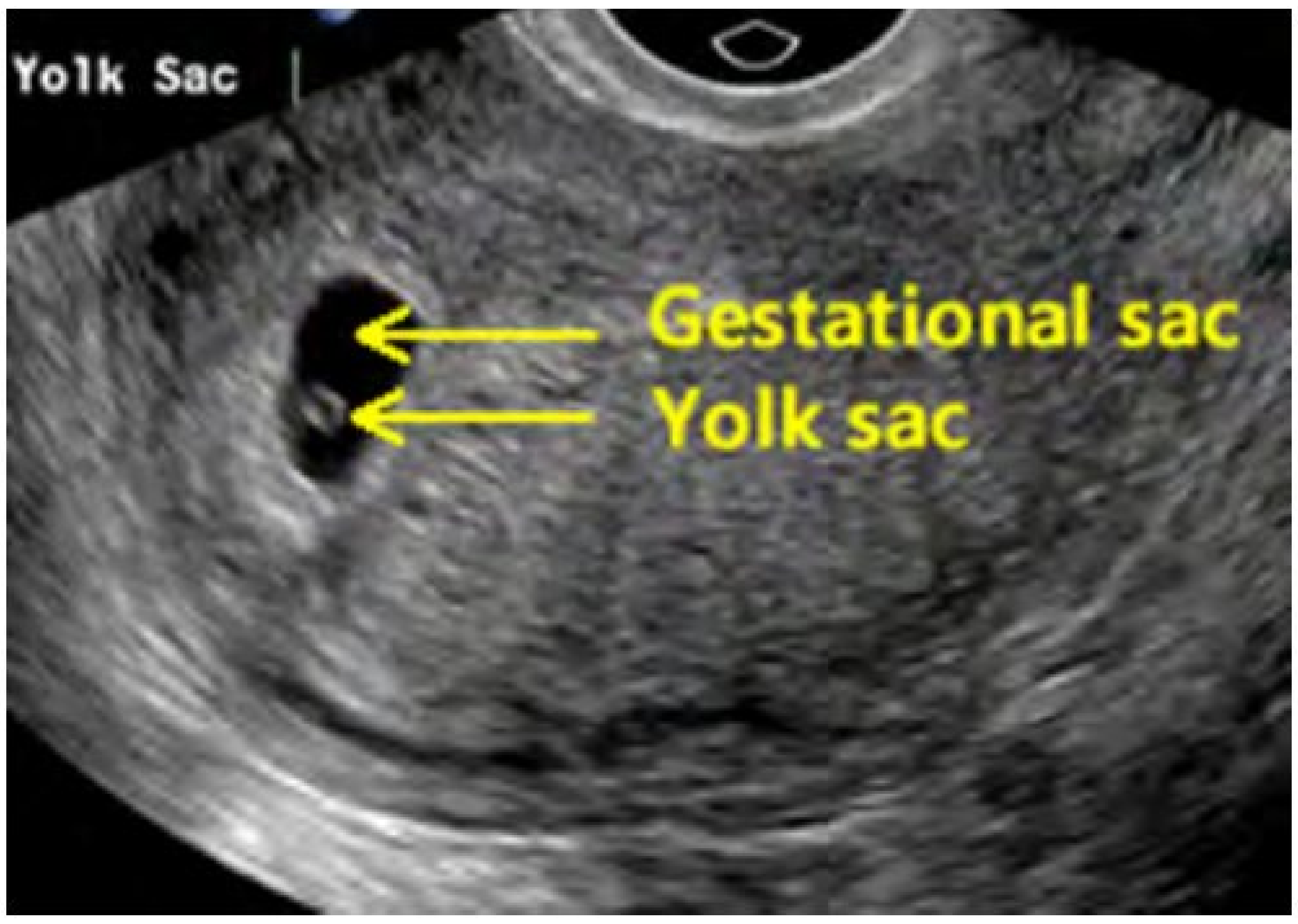
TVS in Early Pregnancy

GESTATIONAL SAC 1st USG Evidence

- it is fluid filled symmetrical structure

Intra-decidual sign - 1st Sign

- s/o Intrauterine pregnancy
- seen at 4.5 - 5 wks

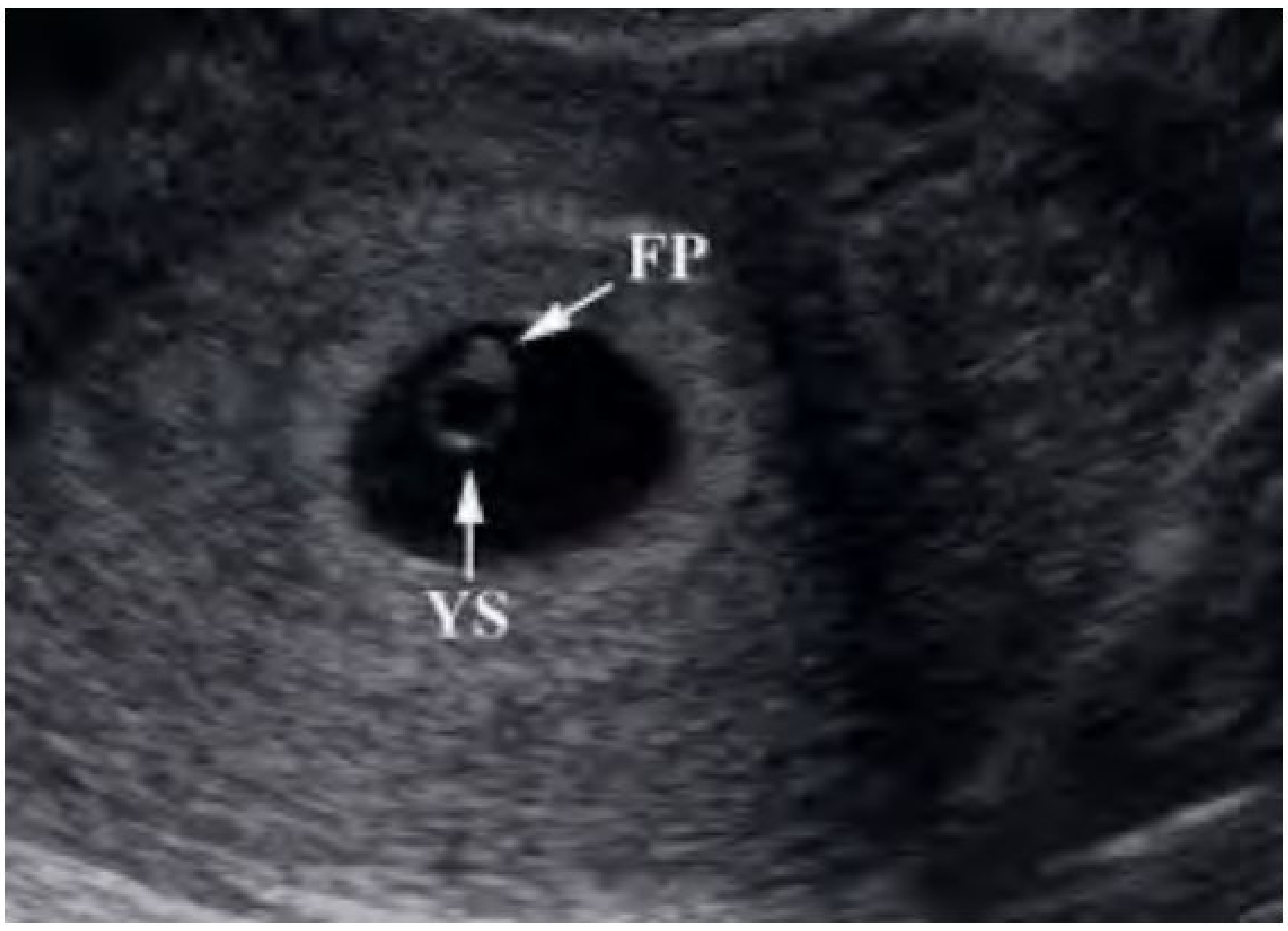


Yolk sac (YS)

- seen between 5-5.5wks
- visible upto 10wks

Fetal Pole (FP)

- cardiac activity seen at 5.5 - 6wks



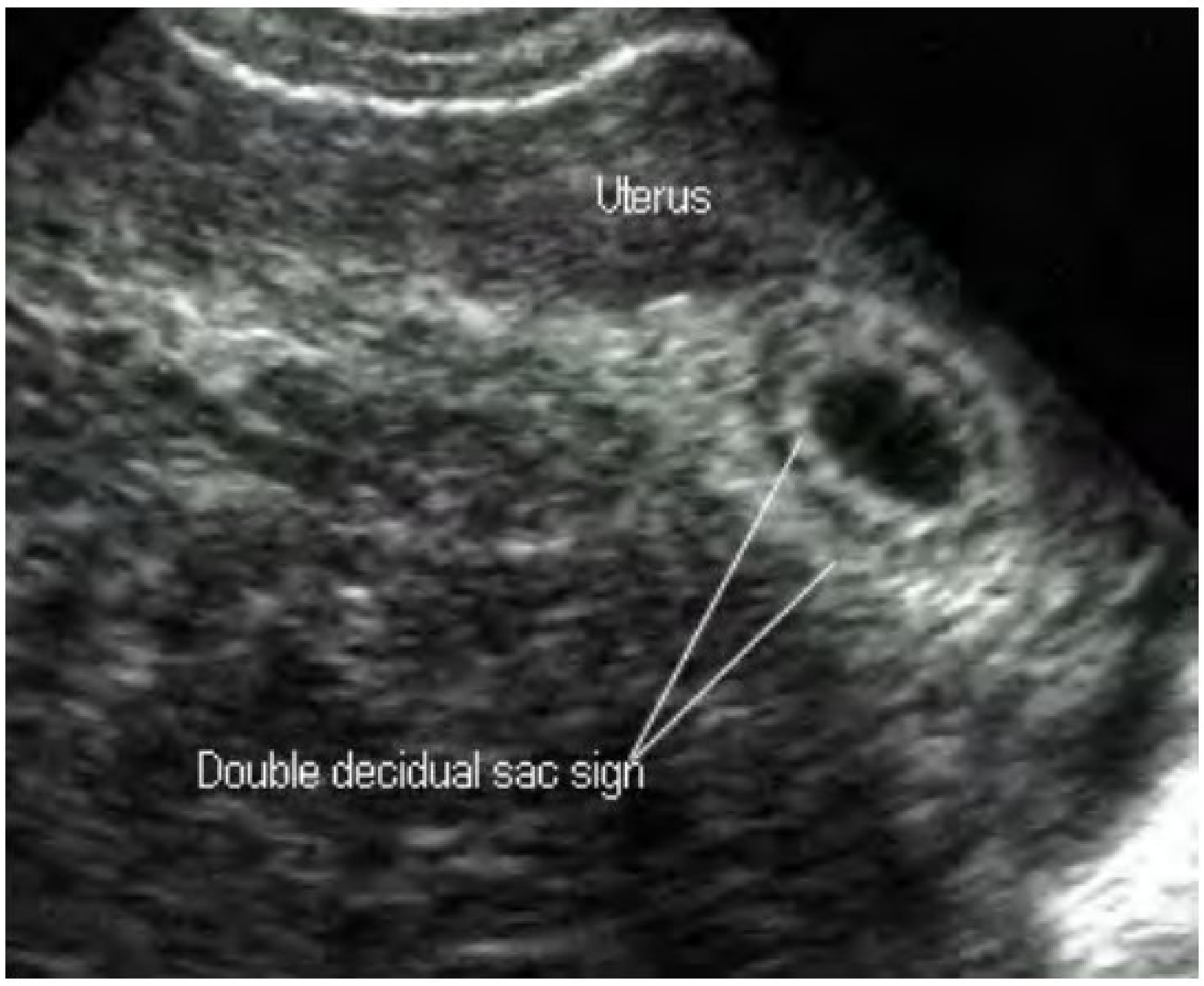
Double Decidual Sac Sign

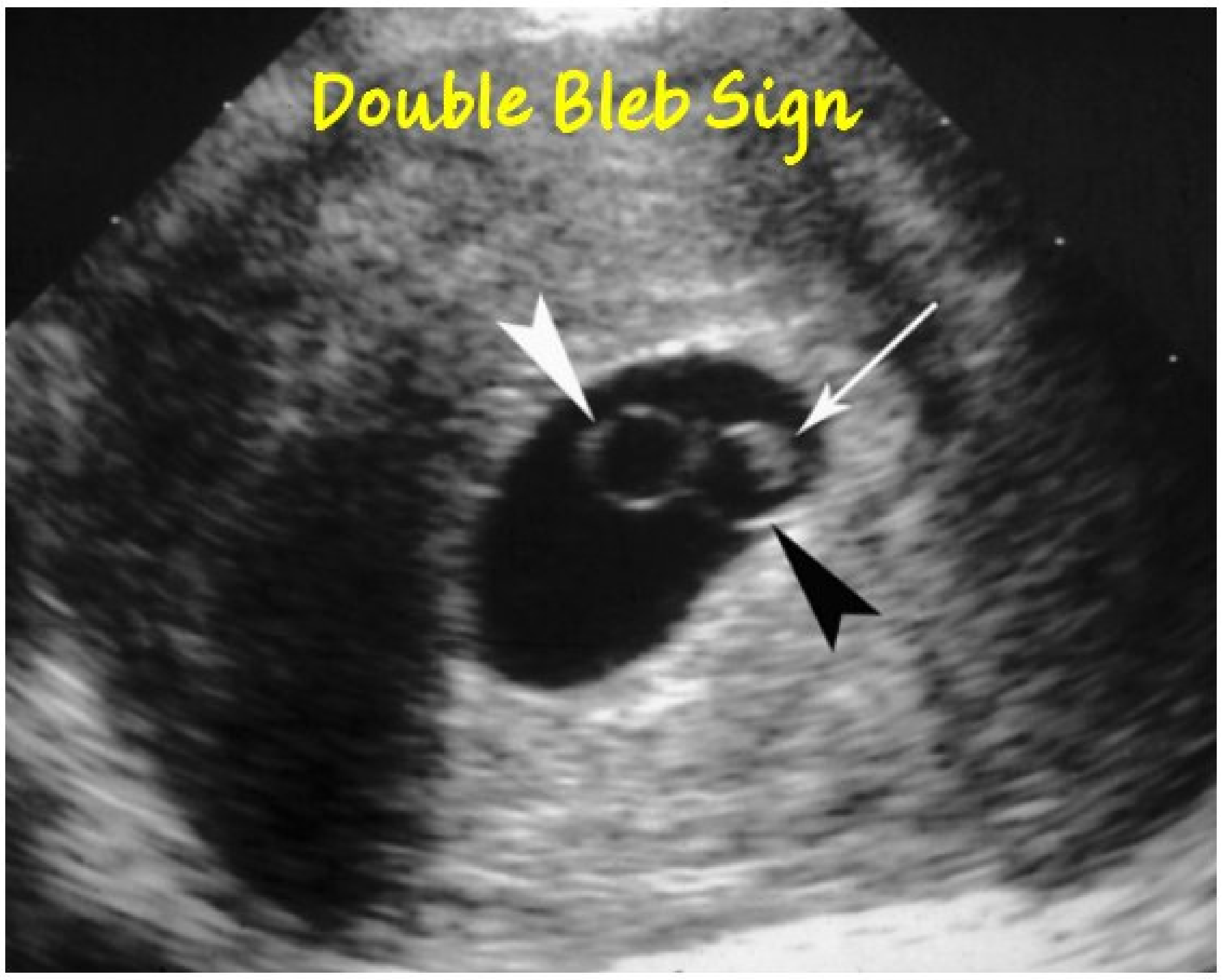
- 2nd early sign

1. Decidua Capsularis
2. Decidua Parietalis

Double Bleb Sign

1. Yolk sac
2. Amniotic sac





cardiac activity on USG – M mode (Motion Mode)

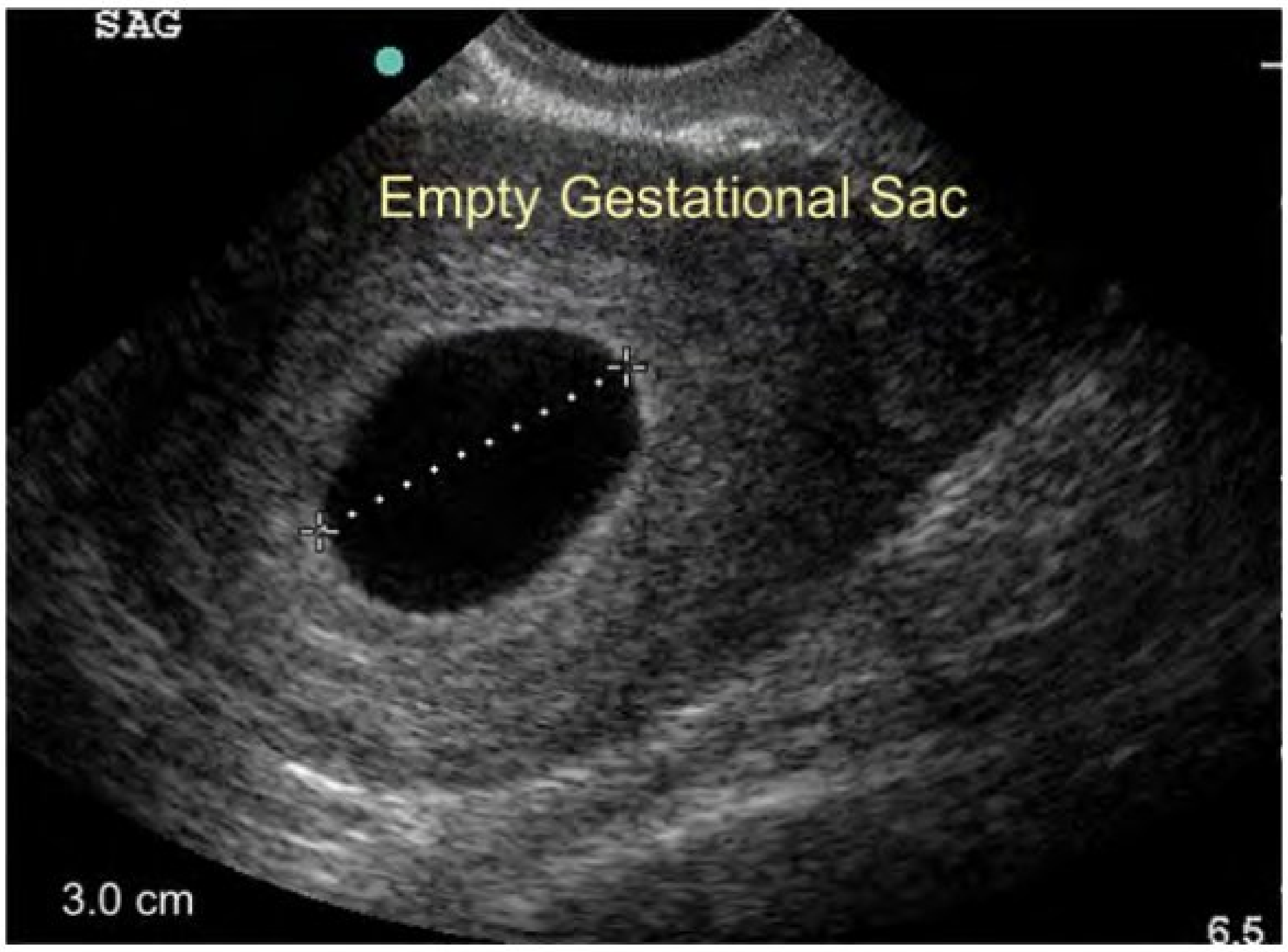
Hand Held Doppler Device – sound of Fetal Heart can be heared as early as 10 wks POG

Viability of Early Pregnancy

- when we see Gestational Sac – measure MSD (Mean Sac Diameter)

BLIGHTED OVUM

- non-viable pregnancy
- if MSD ≥ 25cm but there is no yolk sac/embryo inside
- has avascular villi
- empty sac



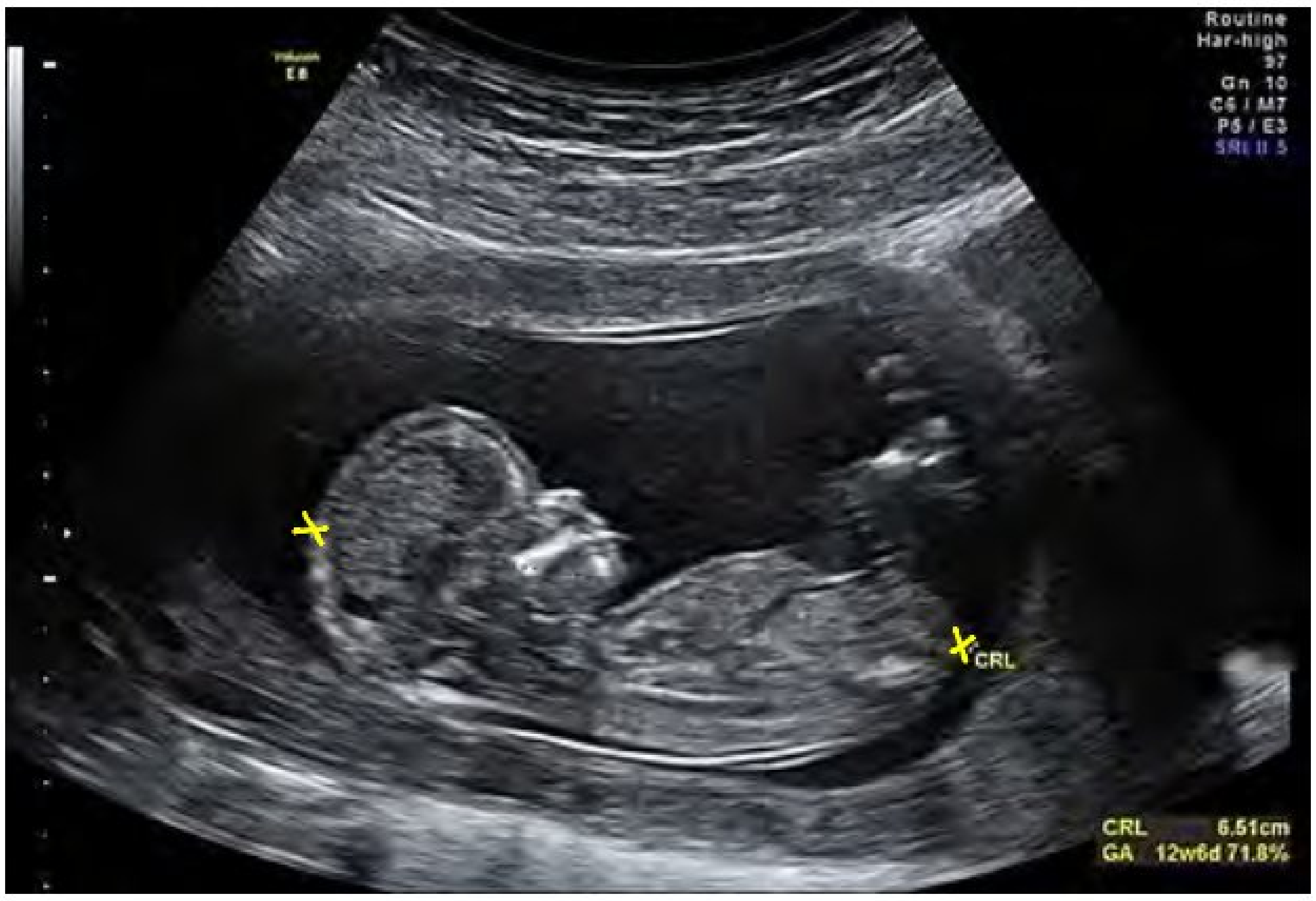
Q. 6wks of amenorrhoea c/o bleeding and pain. On USG MSD of 18mm no yolk sac inside / embryo inside. What's next advice?

Ans: follow up with USG (Repeat USG - minimum after 1wk)

Once we see embryo - measure

CRL Crown Rump Length

- measured from Head end to buttock of baby
- BEST USG parameter to asses Gestational age
- Ideal time to measure between 7 - 10 wks (upto 14 wks)
- if CRL ≥ 7mm c̄ no cardiac activity → Missed Abortion (Non-viable pregnancy)



HCG : Critical value at which Gestational sac should be seen on USG

for TVS - 2000 I.U

for TAS - 6500 I.U

Q. 5wks amenorrhoea c̄ spotting and pain. HCG value 1500 I.U and no Gestational sac seen. What's next advice?

Ans: Repeat HCG after 48hrs

Antenatal Care

POSITIVE OR ABSOLUTE SIGNS OF PREGNANCY

a. Palpation of fetal parts and perception of fetal movements by examiner, at about 20wks

b. Auscultation of fetal heart sounds

c. USG Evidence of embryo (at 6th wk) and later on

d. Radiological demonstration of fetal skeleton at 16wks and onwards.

WEIGHT GAIN IN PREGNANCY

WEIGHT FOR HEIGHT		RECOMMENDED TOTAL WEIGHT GAIN (Kg)
Category	BMI	
Low	<19.8	12.5 - 18
Normal	19.8 - 26	11.5 - 14
High	26 - 29	7 - 11.5
Obese	>29	7

USG IN EARLY PREGNANCY

	TRANSVAGINAL SONOGRAPHY (TVS)	TRANSVAGINAL SONOGRAPHY (TAS)
Gestational sac	4wks 5days	5wks 5days
Yolk sac	5wks	6wks
Fetal pole	6wks	7wks
Fetal cardiac activity	6wks	7wks

Critical titer of hcg to visualize the gestational sac within the uterus:

TVS = 1500

TAS = 3500

BEST PARAMETERS FOR ESTIMATION OF FETAL AGE

1st TM : CRL (Crown Rump Length)

2nd TM : Corrected Biparietal Diameter (cBPD) or Head circumference
Transcerebellar diameter

INFERENCE (HC) 3rd TM : Head circumference and femur length

Overall : CRL (Crown Rump Length)

MENSTRUAL AGE (weeks)	FETAL STRUCTURES
4	Choriodecidual thickness, Chorionic sac
5	Gestation sac, Embryo Yolk sac
6	Fetal pole, Cardiac activity
7	Lower limb buds, Midgut herniation (Physiological)
8	Upper limb buds, Stomach, Internal Gonads formed.
9	Spine, Choroid plexus
10-12 wks	Swallowing Starts
11 wks	Fetal breathing movements
12 wks	External genitalia formed, Urine formation occur

BEST PARAMETERS FOR ESTIMATION OF FETAL AGE

1st TRIMESTER : Crown Rump Length (CRL)

2nd TRIMESTER : Corrected biparietal diameter (cBPD) or
Head circumference trans cerebellar diameter

INFERANCE (HC) 3rd TRIMESTER : Head circumference &
Femur Length

OVERALL : Crown Rump Length

MENSTRUAL AGE (wks)	FETAL STRUCTURES
• 4	• Choriodecidual thickness, Chorionic Sac
• 5	• Gestation Sac, Embryo yolk sac
• 6	• Fetal pole, Cardiac activity
• 7	• Lowerlimb buds, midgut herniation (Physiological)
• 8	• Upper limb buds, stomach, internal gonads formed
• 9	• Spine, choroidal plexus
• 10-12 weeks	• Swallowing starts
• 11 weeks	• Fetal breathing movements
• 12 weeks	• External genitilia formed
• 12 weeks	• Urine formation occurs

EXTRA EDGE : USG IN PREGNANCY

i. Best time to assess gestational age by USG is 9-14 weeks (by Crown Rump Length)

ii. Best indicator of fetal growth, IUGR - Abdominal circumferences.

iii. Transcerebellar Diameter (TCD) is an accurate predictor of gestational age b/w 14-28 weeks

iv. Fetal anomaly which can be earliest detected by USG - Anencephaly (Level I)

v. Lemon & Banan Sign are seen in Neural Tube Defects c̄ spinal involvement

vi. Two Best ultrasonographic markers of Down Syndrome in Ist Trimester
→ Absent or Hypoplastic nasal bone
→ Increased nuchal translucency more than 3.5 mm

vii. CRL (in mm) + 42 = Gestation in Days

viii. Embryo should increase its CRL by 1mm per day. Failure to visualize an embryo when the MSD is 20mm, indicates pregnancy loss. It indicated blighted ovum which is most commonly because of Chromosomal Anomaly

ix. Doppler to be avoided in Ist Trimester. It leads to thermal inj. to fetus.

SIGNS OF PREGNANCY IN 2nd & 3rd TRIMESTER

PHENOMENON	TIME
· Palpation of fetal part	20 weeks
· Active fetal movement by placing a hand on Abdomen	20 weeks
· External Ballotment	20 weeks
· Internal Ballotment	16-28 weeks
· FHS Audible by Stethoscope	18-20 weeks
· Fetal movement can be detected by Doppler	10 weeks
· Lightening	38 weeks

ANC VISITS

IDEALLY THE SCHEDULE FOR ANTENATAL VISITS SHOULD BE:

a. Monthly visits upto 28 weeks
b. Two weekly visits b/w 28 & 36 weeks
c. Weekly visit from 36 weeks onwards.

} Total 12-15 visits

WHO RECOMMENDS AT LEST 4 VISITS:

1st visit : 16 weeks
2nd visit : 24-48 weeks
3rd visit : 32 weeks
4th visit : 36 weeks

At each visit do:
- HGM
- Sugar
- BP
- Wt gain assessment
- Urine Albumin

AS PER INDIAN SCENARIO- MINIMUM 3 VISITS -ESSENTIAL

1st at : 20 weeks (or as soon as pregnancy is known)
2nd at : 32 weeks
3rd at : 36 weeks

First visit that a woman makes to health care facility : BOOKING VISIT

A booked case is one that has at least 3 antenatal visits with at least two in last trimester

INVESTIGATION

a. Blood : HB, BT, CT, BG, VDRL, HBsAg, HCV, HIV.
BG - FASTING & POST PRANDIAL, HPLC

b. Urine complete

c. Double marker at 11-13+6 weeks

d. Triple marker (MSAFP) - 15-18 weeks

e. NT NB Scan at 11-13+6 weeks

f. Level II Scan (18-20 weeks)

KEGELS EXERCISES - TIME FOR INITIATING KEGELS EXERCISE

a. Pregnancy - 3rd Trimester

b. After vaginal delivery : after 24 hrs

c. After cesarean section : after 24 hrs

VACCINES IN PREGNANCY

killed vaccines are safe in pregnancy

Live vaccines are best avoided in pregnancy.

SAFE	ONLY IN EPIDEMICS	To be given in case of travel to highly endemic areas or exposed to contacts	C/I
H: Hepatitis A or B I: Influenza T: Tetanus Rabies	Tab : Typhoid P : Pneumococcus C : Cholera M : Meningococcus	Yellow Fever Japanese encephalitis Polio (IPV)	Rubella Measles Varicella BCG Mumps Smallpox

High Risk Pregnancy: A high risk pregnancy is one where the mother suffers from causes which are detrimental to her health or fetal health. Some of these important factors are :

i. Age >35 yrs
ii. Elderly primi
iii. >5 previous pregnancies
iv. H/O Past still births
v. H/O Neonatal births
vi. Previous caesarian section
vii. Hemorrhage
viii. Placental previa
ix. Short stature - below 140 cm
x. Young primi - <19 years
xi. PET
xii. Systemic Diseases -
HTN, RHD, Diabetes
, Renal diseases etc.
xiii. TB

PRE-CONCEPTIONAL & ANTEPARTUM SCREENING & COUNCELLING

- Pre conceptional screening & councelling offers an opportunity to identify & mitigate maternal risk factors before pregnancy begins.
- The preconceptional visit is the single most important health care visit when viewed in the context of it's effect on pregnancy
- Barker Hypothesis : The intra-uterine fetal enviornmental has a tremendous impact on the health & well-being of the adult that fetus will become. (IUGR babies are more prone to develop coronary artery disease, HTN, Diabetes, Dementia in future)

PRENATAL DIAGNOSIS

- Prenatal diagnosis employs a variety of technique to determine the health & condition of an uunborn fetus.
- Congenital anomalies account for 20-25% of perinatal death.
- The techniques employed for prenatal diagnosis include

CAUSES OF CONGENITAL ABNORMALITIES

- CHROMOSOMAL (6%)
  - Trisomy - 21 (Down synd.)
  - Trisomy - 18 (Edward's synd.)
  - Trisomy - 13 (Patau's Synd.)
- SINGLE GENE DISORDER (5%)
  - AUTOSOMAL
    - DOMINANT (70%)
      - Achondroplasia
      - Marfan Syndrome
      - Neurofibromatosis
    - RECESSIVE (20%)
      - Cystic Fibrosis
      - Galactosemia
      - Sickle cell anemia
  - XL Recessive - 5%
  - XL Dominant - Rare
    - Hemophilia
    - Duchenne muscular dystrophy
    - Clour blindness
    - Fragile X Syndrome
- INFECTIONS (2%)
  - Rubella
  - CMV
  - Varicella
  - Parvo virus
  - Toxoplasma
- DRUGS & ENVIORNMENTAL (1-2%)
  - Warfarin
  - Lithium
  - Dilantin
    - * Radiation
    - * Alcohol
    - * Hypoxia

OTHER CAUSES

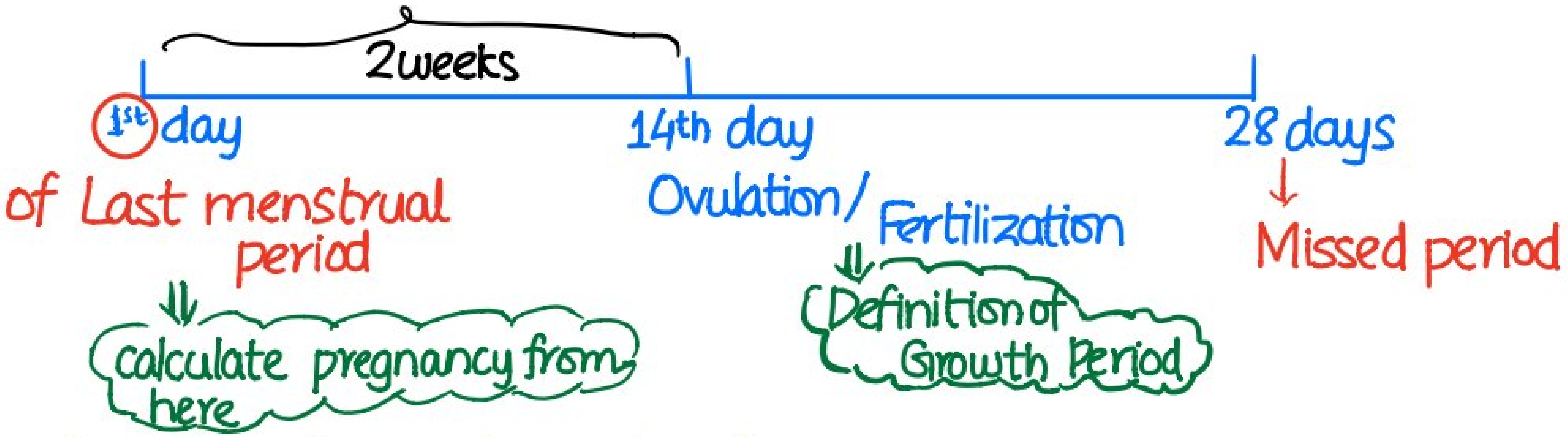
a) MATERNAL ILLNESS (5%)
- Diabetes
- Epilepsy

b) MULTIFACTORIAL (20%)
- Neural Tube Defects
- Congenital Heart defects
- Cleft Lip & Palate

c) IDIOPATHIC (60%)

Fetus

GESTATIONAL AGE / FETAL AGE / PERIOD OF GESTATION


2weeks
1st day
of Last menstrual period
calculate pregnancy from here
14th day
Ovulation / Fertilization
Definition of Growth Period
28 days
Missed period

One day of missed period = 4 weeks pregnant

* Event : x days from day of fertilization

That event = x days + 2 weeks of Pregnancy

Eg : Trophoblast divides into Cytotrophoblast & Syncitiotrophoblast 8 days after fertilization

at what gestational age trophoblast divides ?

2wks + 8 days

* Any event which occurs anytime from period of gestation /at pregnancy ⇒ What time after fertilization it is going to happen ? ⇒ Substract 2 wks from it

Eg : Cardiac activity is seen in fetus at 5 wks of pregnancy

After how many weeks after fertilization, is cardiac activity seen ??

⇓

5 wks - 2 wks = 3 wks after fertilization

FETAL GROWTH PERIOD

Are defined from the day of fertilization and not from the 1st day of LMP

Growth Period	Normal Definitions (from day of fertilization)	Definition as per period of Pregnancy(+2)
Fetilized ova	Day of fertilization uptill 2wks of Pregnancy	uptill 4 wks of 4 weeks of Pregnancy
Embryonic Period Teratogenic	From 3wks after fertilization and uptill 8 wks after fertilization	From 5wks uptill 10 wks of pregnancy
Fatal Period	≥ 9wks after fertilization & uptill delivery	11 wks of pregnancy & uptill delivery

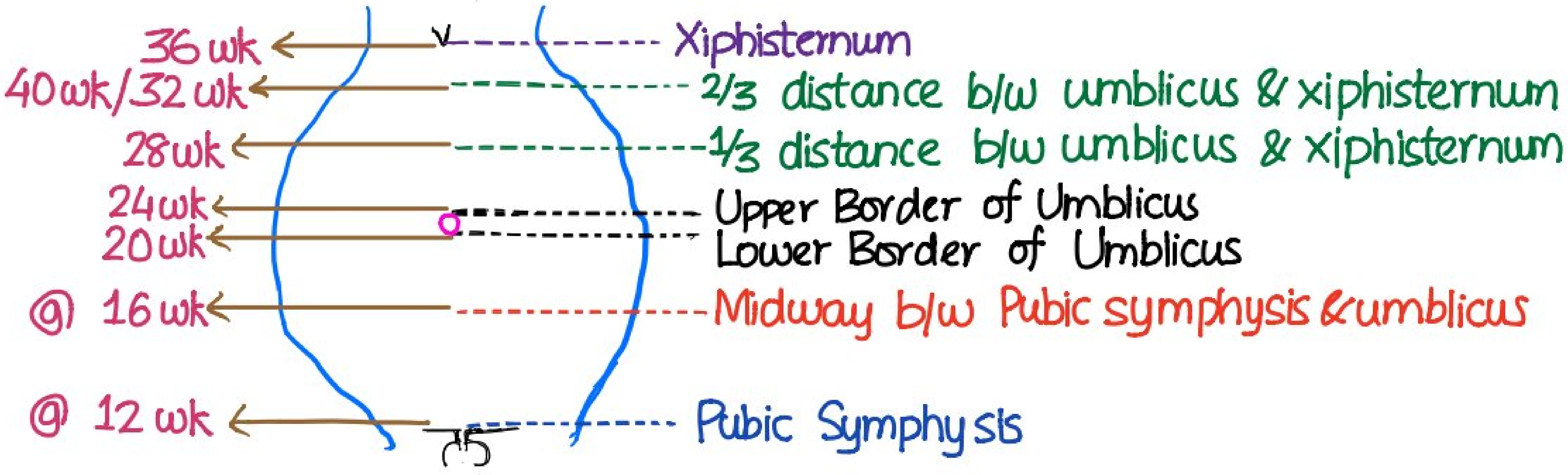
THE TOTAL DURATION OF PREGNANCY

280 Days / 40wks / 9month + 7days

- 1st trimester ⇓ uptill 13 wks + 6 days
- 2nd Trimester ⇓ 14 wks → 27wks + 6days
- 3rd Trimester ⇓ 28wks → 40wks

ESTIMATING FETAL AGE

CLINICALLY


36 wk ← Xiphisternum
40wk/32 wk ← 2/3 distance b/w umblicus & xiphisternum
28wk ← 1/3 distance b/w umblicus & xiphisternum
24wk ← Upper Border of Umblicus
20wk ← Lower Border of Umblicus
@ 16 wk ← Midway b/w Pubic symphysis & umblicus
@ 12 wk ← Pubic Symphysis

After 36 wks → Head of fetus goes down into pelvis
⇓
Height of Uterus ↓
↓
Respiratory Discomfort ↓
↓
K/a Lightening / Welcome sign

How to know whether it's 32 wks / 40 wks of Pregnancy??

If fetal head is felt per abdomen = 30 wks
If fetal head is not felt per abdomen = 40 wks

ON USG : ESTIMATION OF FETAL AGE

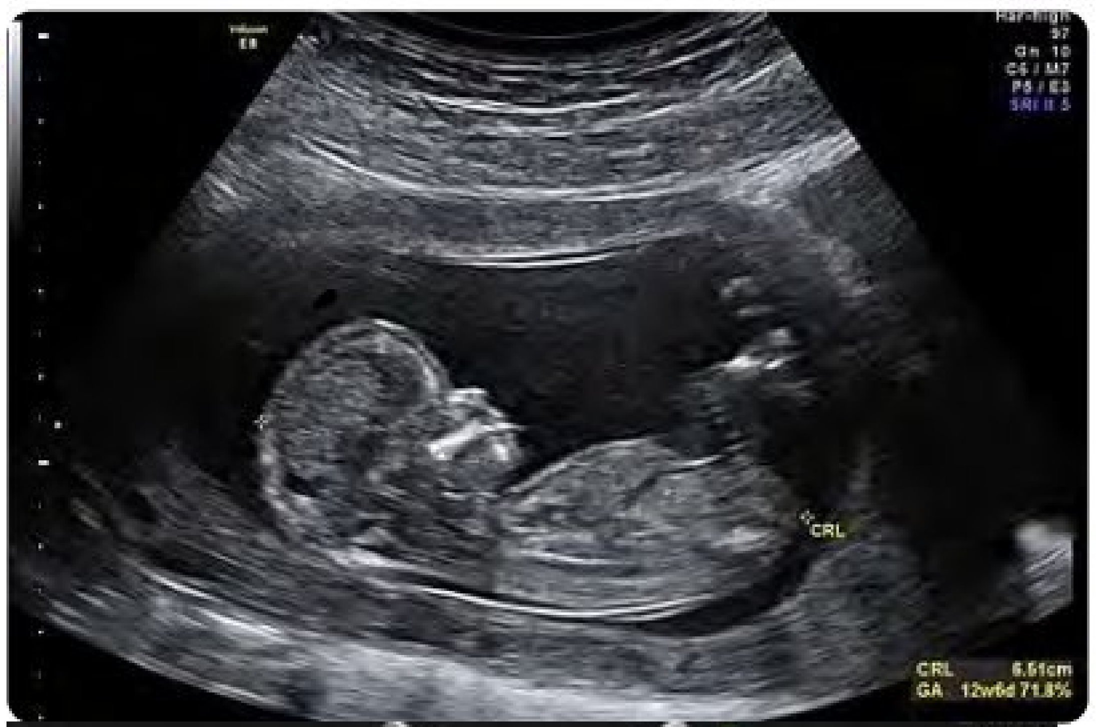
TRIMESTER	BEST PARAMETER
1st TRIMESTER	Crown Ramp Length
2nd TRIMESTER	Best : Biparietal diameter (BPD 2nd : Head Circumference
3rd TRIMESTER	Femur Length

IMPORTANT ASPECTS

1. The earlier the USG done in pregnancy, the more accurately it predicts Fetal age
2. Suboptimally dated on USG → If a patient has not got any USG done before 22 wks of pregnancy.
3. EDD should not be changed based on USG except in cases of IVF

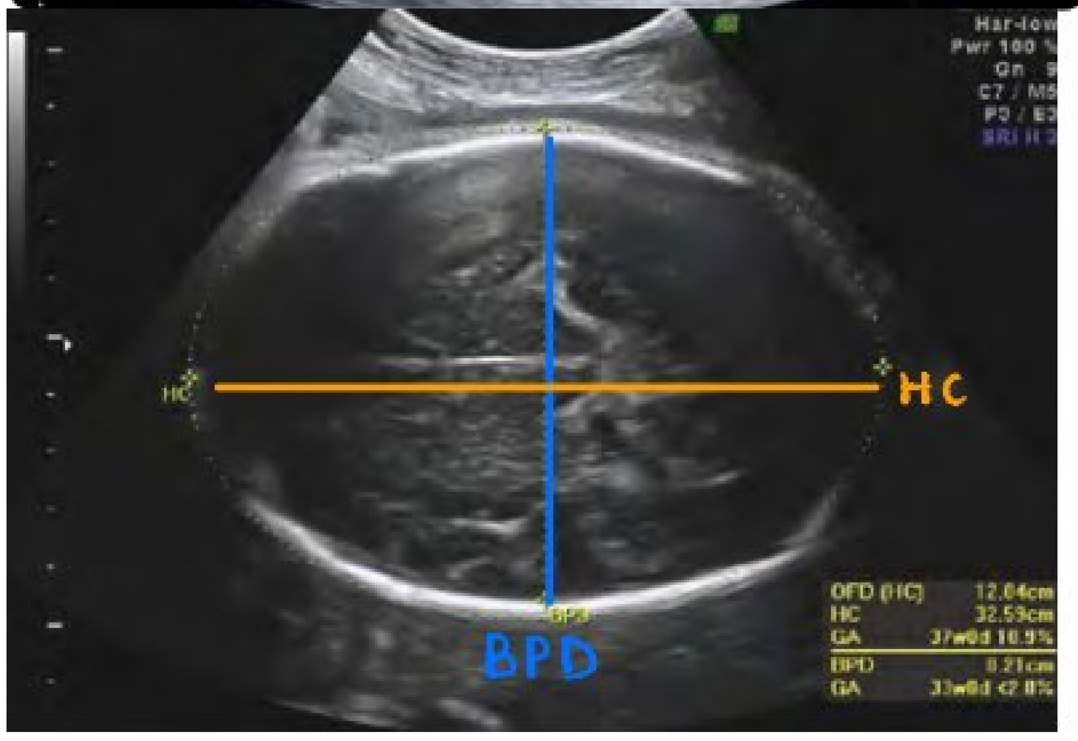
Important Points on CRL (Distance b/w Vertex & Coccyx)

1. CRL + 6.5mm = Gestational age in weeks
   Eg: Suppose, CRL is 8mm, add 6.5mm to CRL = 14.5 wks
2. CRL + 42 = Gestational age in days
3. CRL ↑ by 1mm/day
4. Smallest CRL at which embryo is viable = 4-5mm
5. If CRL ≥7mm + No cardiac Activity
   ⇓
   Missed Abortion



Important points on BPD

It measured from,
Outer table of fetal skull on the one side to inner table of fetal skull on other side.



Fetal age can also be estimated by ; HASSE RULE

<5 months of Pregnancy
↓
$\sqrt{\text{fetal length}}$
↓
Gestational Age

Fetal length ⇒ vortex to coccyx
↓
CROWN RUMP LENGTH

≥5 month of Pregnancy
↓
$\frac{\text{Fetal Length}}{5}$
↓
Gestational Age

Fetal length ⇒ vertex to heel of fetus
↓
CROWN HEEL LENGTH

ASSESMENT OF WEIGHT OF FETUS

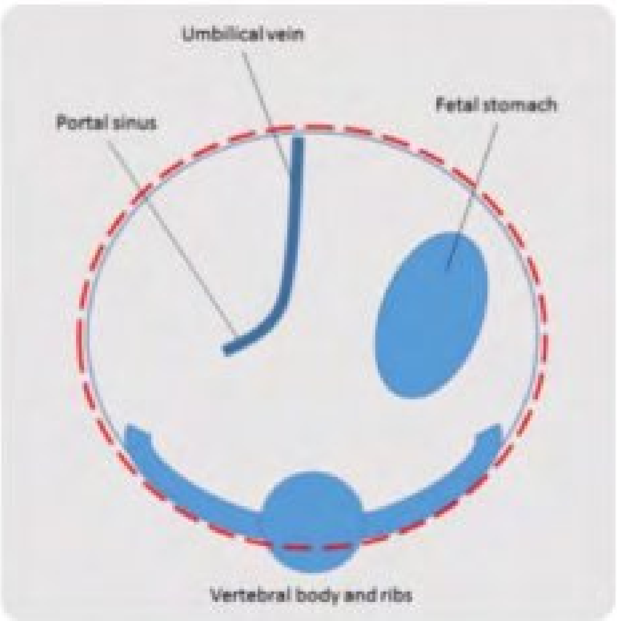
Clinically
↓
JHONSON FORMULA

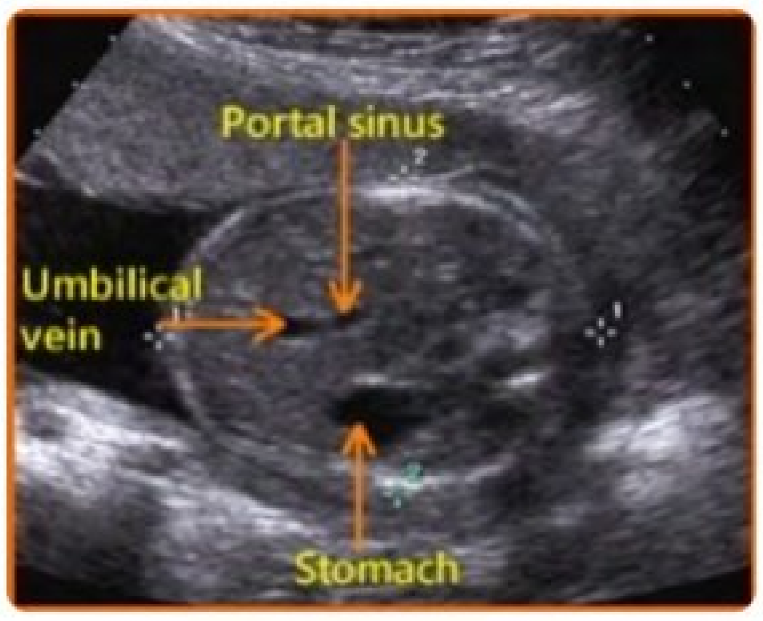
USG
↓
HADLOCK FORMULA
SCHEPHERD FORMULA

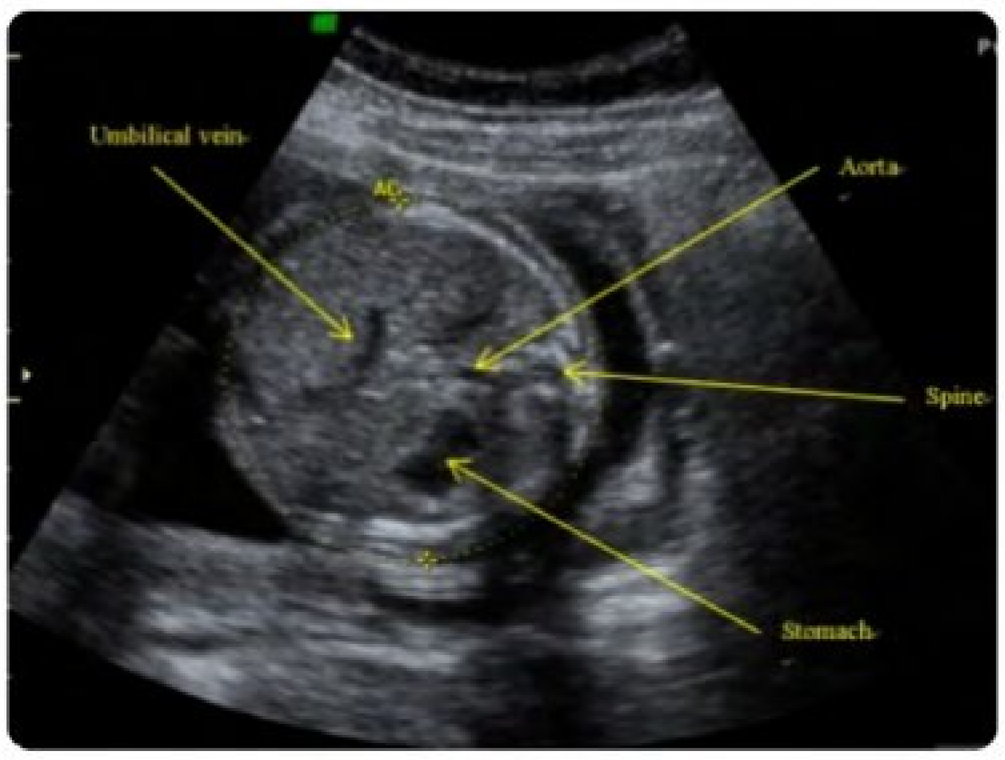
ASSESMENT OF FETAL GROWTH

Only 1 USG Parameter
↓
ABDOMINAL CIRCUMFERENCE

Abdominal Circumference is measured in a plane where following are visible → Portal Sinus
Umblical veins
Stomach







Abdominal circumference is the only parameter which is telling about growth of fetus

↙ IUGR ↘ Macrosomia

If AC ≥ 35cm ⇒ Macrosomia

Normally = FL/AC = 20-40%

Ratio increased → IUGR

Ratio decreased → Skeletal Dysplasia

FETAL HEMATOEISIS

SITE	TIME OF PREGNANCY	MAIN Hb FORMED
·Yolk Sac →	6 wks of Pregnancy →	GOWER 1 GOWER 2 PORTLAND Hb
·Liver →	Upto 20 wks →	HbF
·Bone marrow →	After 20 wks →	Hb A

FETAL ERYTHROCYTES (RBC)

In Early Intrauterine Life ⇒ Fetal RBC

↙ Nucleated ↘ Macrocytic (MCV = 180 FL)

↓ Lateron size ↓ = 105 FL

{But this not happen in Aneuploid Fetus}

At time of birth = Hb ; 16-18 gm/dl

75-85% ⇓ HbF

20-25% ⇓ HbA

But by 6 months of age, HbF is <1%

HbF	HbA
-Less of 2,3-DPG	-More
-Less carbonic anhydrase	-More
-Higher affinity for O_2	-Low affinity
-Resistant to acid & alkali	-sensitive to acid & alkali

⇓

Forms the basis of 2 tests

1. API TEST/SINGERS alkali denaturation test
2. Kleihaver Betke test

APT TEST (or) SINGER'S ALKALI DENATURATION TEST	KLEIHAVER BETKE TEST
* Helps to differentiate b/w placenta previa & vasa previa	* Calculate the dose of Anti-D in Rh -ve females
* 1% NaOH or KOH Reagent	* Citric acid phosphate Buffer Reagent
* Qualitative test	* Quantitative test
* Differentiating b/w fetal blood & maternal blood.	* Differentiating b/w fetal RBC & maternal RBC

MAJOR EVENTS IN FETAL DEVELOPMENT

EVENTS	SEEN AT
Fetal gross body movement	7 wks
Gonads formation (T>ovaries)	7 wks
Glucagon synthesis	8 wks
Internal genitalia	10 wks
Breathing movements (M&F)	11 wks
External Genitalia Female Male	12 wks 14 wks

100% sex of fetus on USG determined by 14 wks	
Insulin production	12 wks
HP axis	12 wks
Fetal urine	12 wks (3 months)
Meconium production	16 wks (4 months)

NOTE: * Fetus produces only IgM: Begins at 20 wks
IgM & Albumin cannot cross placenta
IgG is transported to fetus at 16 wks
* Surfactant production begins at 20 wks
↳ Present in Amniotic Fluid at 28 weeks

USES OF USG IN PREGNANCY

To confirm pregnancy :

SIGNS	TVS	TAS
Gestational Sac	4 wks + 1 day to 4 wks + 3 day	5 wks
Yolk Sac	5 wks	5.5 wks
Cardiac activity	5-6 wks	6-7 wks

Q Earliest time at which Gestational sac can be identified on TVS from LMP?

a) 20 day
b) 25 day
[✓ c) 30 day]
d) 35 day

4 wks + 1 day = 29
to
4 wks + 3 day = 31
28+3

Q Earliest time at which gestational sac be identified on TVS from fertilization ?

[✓ a) 20 days]   c) 30 days
b) 25 days   d) 35 days

2 wks + 1 day = ⑮
to
2wks + 3 day = ⑰

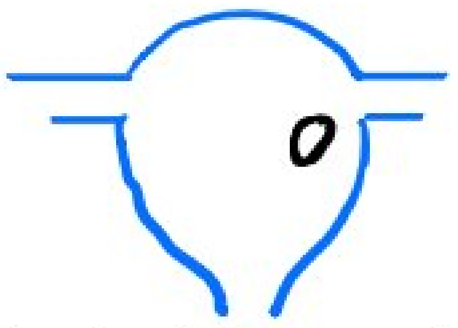
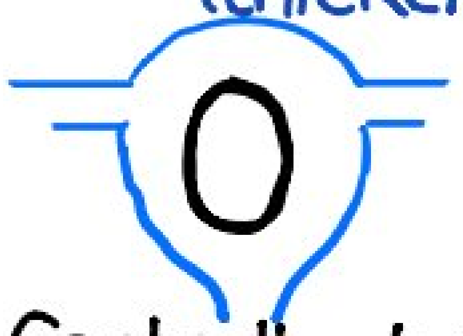
Any event which is happening from LMP & we have calculate it from day fertilization
↓
Substracte 2 weeks

Q Gestational sac can be visualized on USG :

a) 4 wks   c) 6 wks
[✓ b) 5 wks]   d) 7 wks

Unless it is not mentioned whether TVS (or) TAS
⇓
Take it as TAS

2. It helps in knowing whether it's Intrauterine or Ectopic Pregnancy

INTRAUTERINE PREGNANCY	ECTOPIC PREGNANCY
True Gestational Sac  * Located Eccentric * Grows (↑ in size) Everyday * Double decidual sac present.	Pseudo gestational Sac (thickened decidua)  * Centrally located * Doesnot grows * Double decidual sac absent.

* 1st sign of pregnancy on USG = Gestational Sac
* 1st sign of intrauterine pregnancy on USG = Yolk sac

1. Suppose :- Gestational sac = Mean sac diameter ≥ 25mm & no fatal tissues / Yolk sac / No cardiac activity
⇓
BLIGHTED OVUM

2. If CRL ≥ 7mm but no cardiac activity
⇓
MISSED ABORTION

CASE SCENARIO

Female with 6wks ammonorhoea + Spotting present
On USG = Mean sac diameter = 20mm
⇓
NEXT STEP ?
→ wait for week & then repeat USG

3. Helps in diagnosing twin pregnancy
4. Help to know Fetal Age
5. Helps in assesing growth of fetus
6. Helps in detecting congenital malformation

CONGENITAL MALFORMATIONS & USG

Routine USG
↓
Type 1 USG
↓
Can detect only few Congenital Malformations
↓
One that congenital malformation is ANENCEPHALY

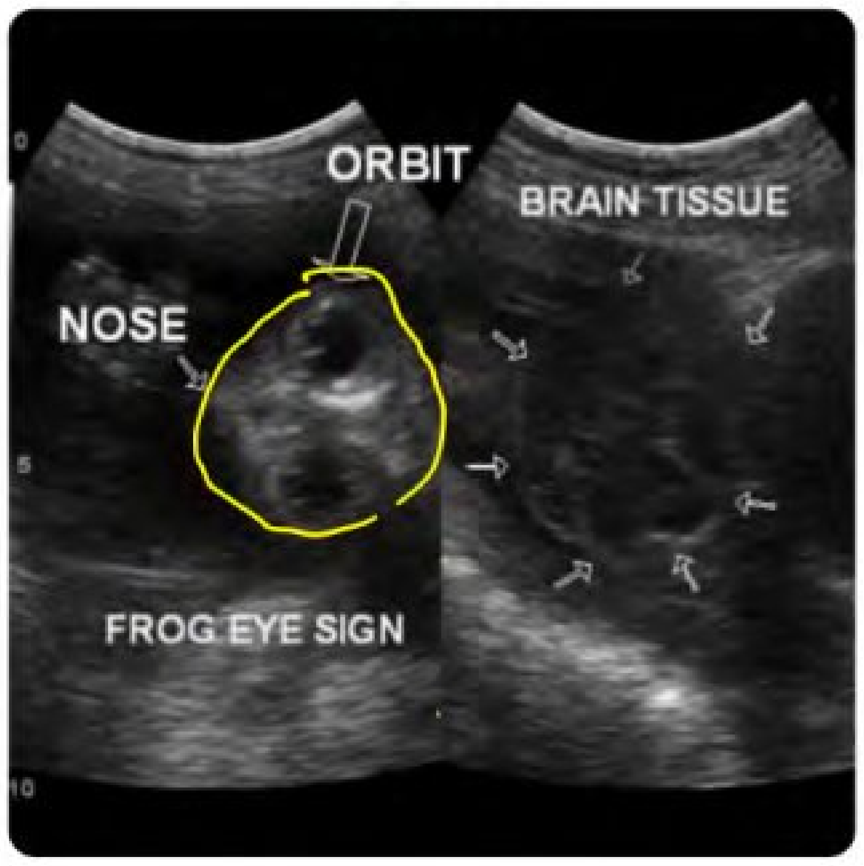
TYPE 2 USG / TIFFA
Targetted Imaging For Fetal Anomalies
⇓
Can detect all congenital malformations
⇓
Done in all pregnant females
Best time = 16-20 wks (18-20 wks)
(Precisely)
↓
Why ?
⇓
1. All organogenesis completed by 20 wks
2. In INDIA MTP is legal upto 2 weeks

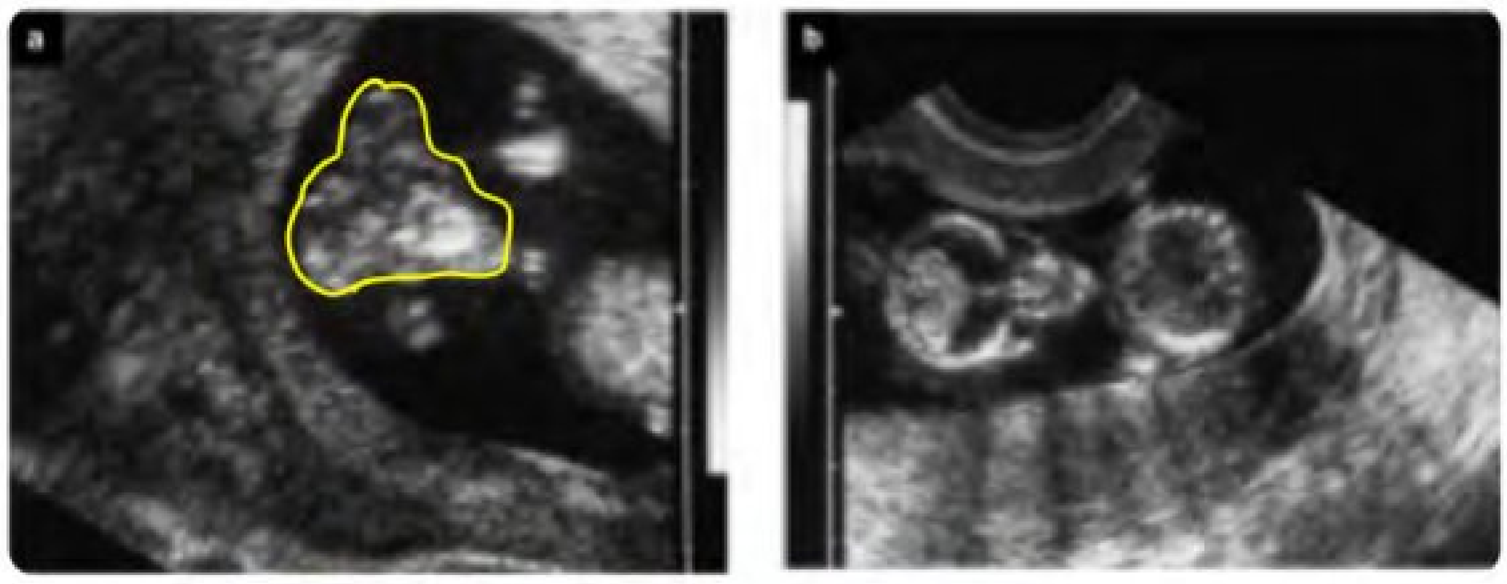
During entire pregnancy, only once USG has to be done, which USG should be that one & what time it should be done ??

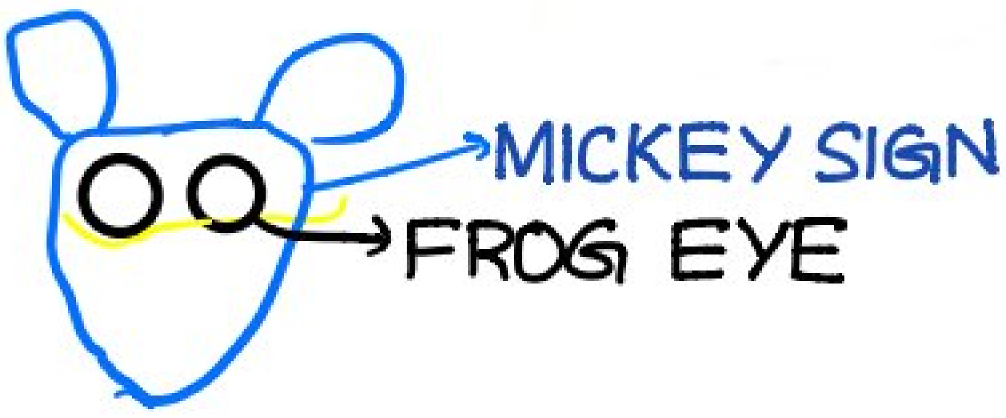
TYPE 2/TIFFA @ 16-20 wks
(18-20wks)

ANENCEPHALY & USG

- Earliest time detected on USG
  - 10 wks
- Best time
  - 14 weeks
- Signs
  - i. Frog Eye Sign
  - ii. Mickey Mouse sign

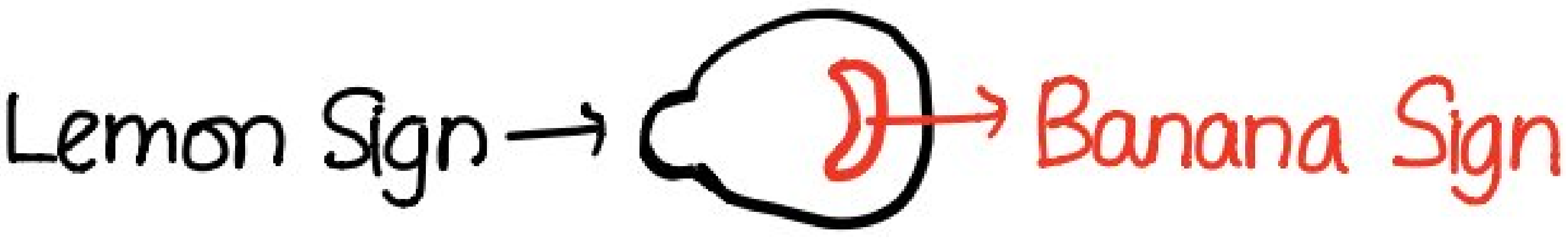


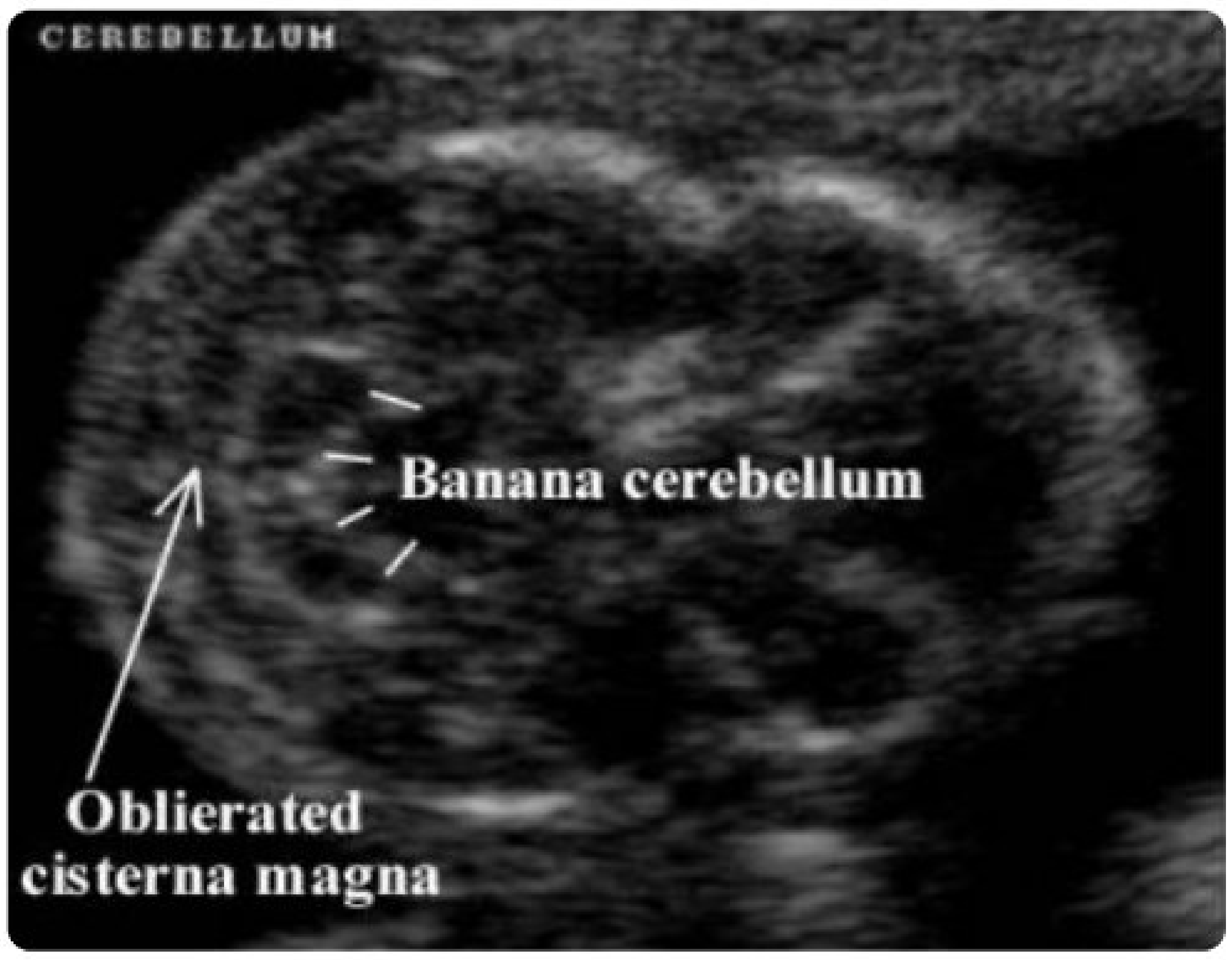




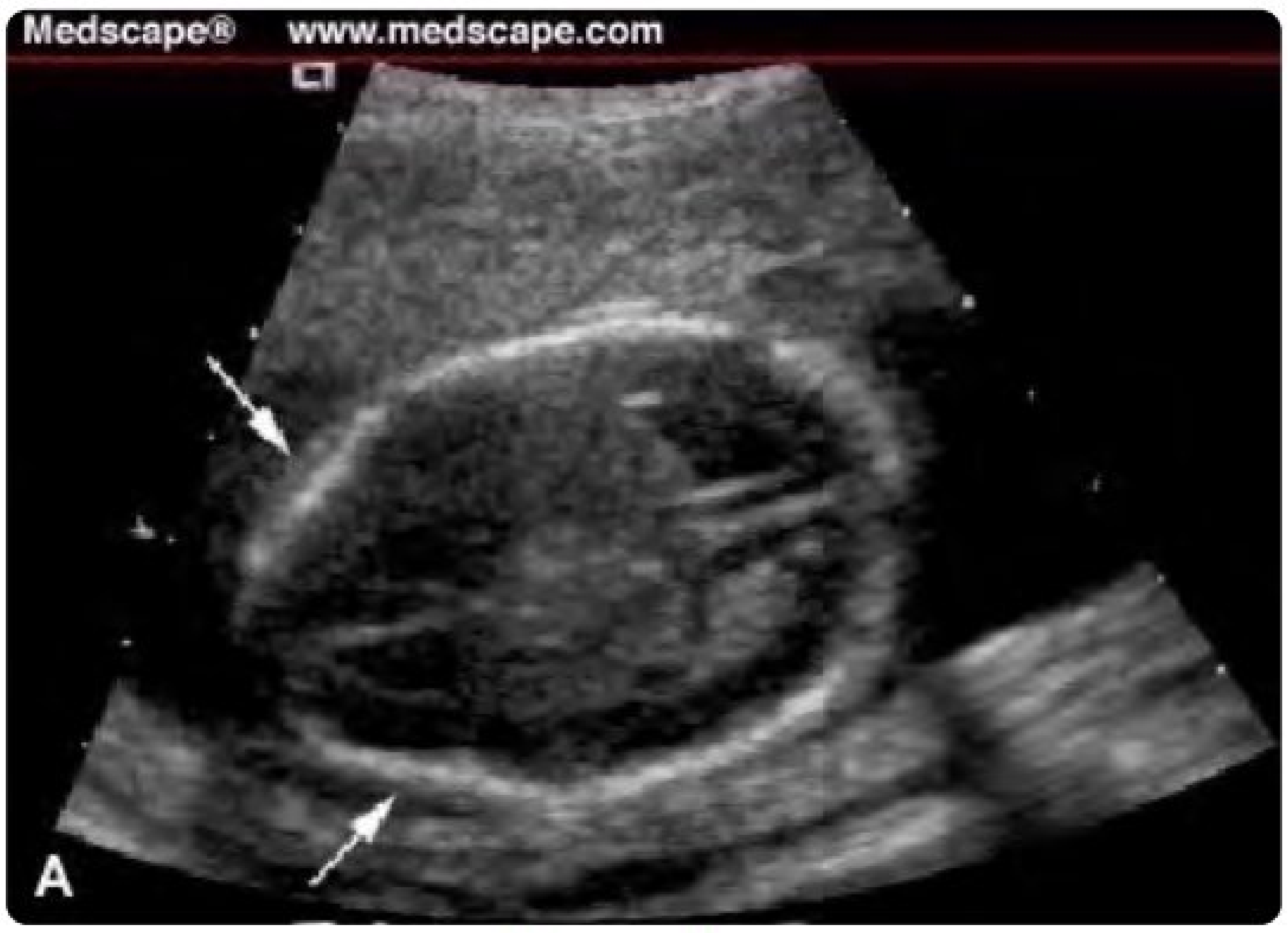
USG SIGNS INDICATING SPINA BIFIDA

- Small Biparieta diameter
- Ventriculomegaly
- Frontal bone scalloping (the so called Lemon sign)
- Elongation & downward displacement of the cerebellum (the so called Banana sign)





Banana sign



Lemon sign

ANOTHER DEFECT WHICH CAN BE DETECTED IN 1st TRIMESTER

are ventral wall defects

↓

i. Omphalocele → Bowel contained in sac & there will be covering over Bowel

ii. Gastroschisis → There is no covering over Bowel

We know that, there is physiologic Herniation of Intestine which happens

If we are getting ventral defects before 12 wks of pregnancy.

↓

- Defect continuing in 2nd trimester USG
  - ↓ Omphalocele (or) Gastroschisis
- Physiologic Herniation of Intestine

If size of defect is ≥7mm + Defect continoues even after 12 wks of pregnancy

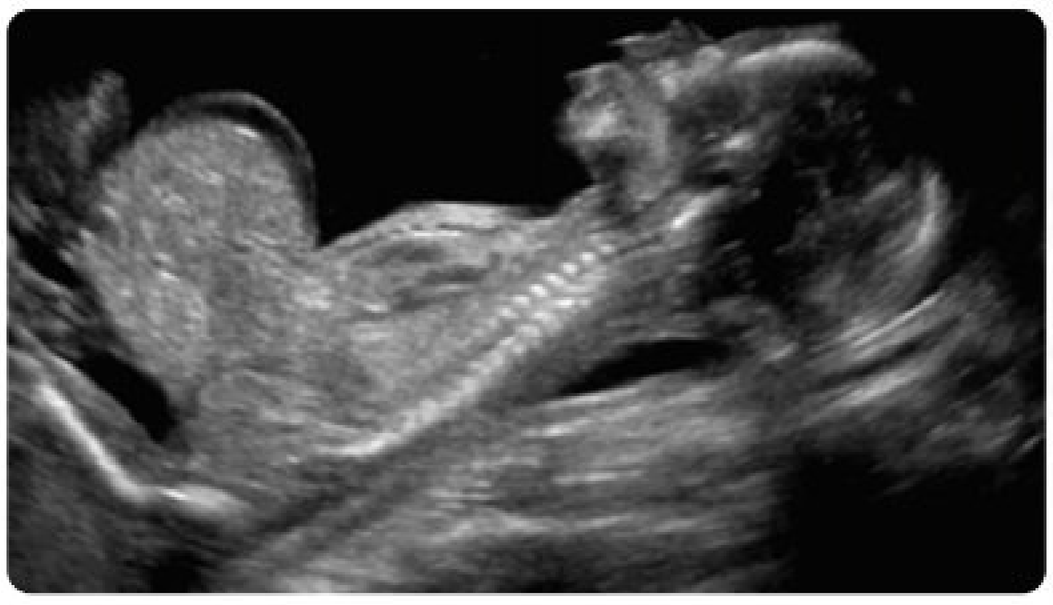
⇓

Pathological

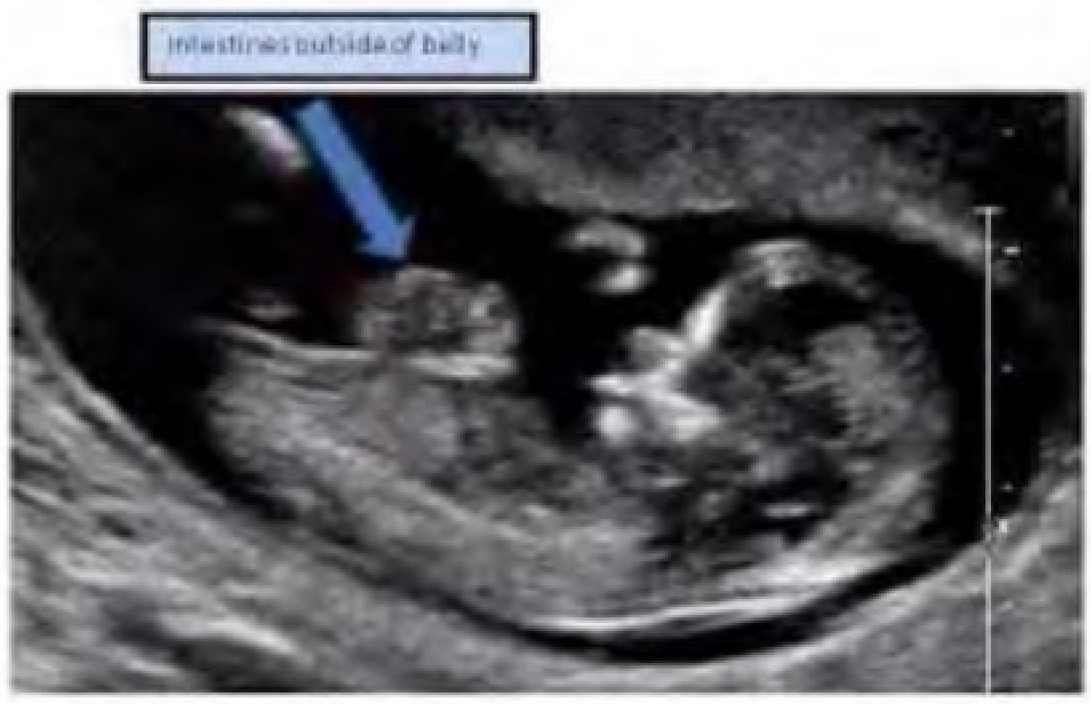
- OMPHALOCELE
- GASTROSCHISIS

Omphalocele → Defect is smooth contour

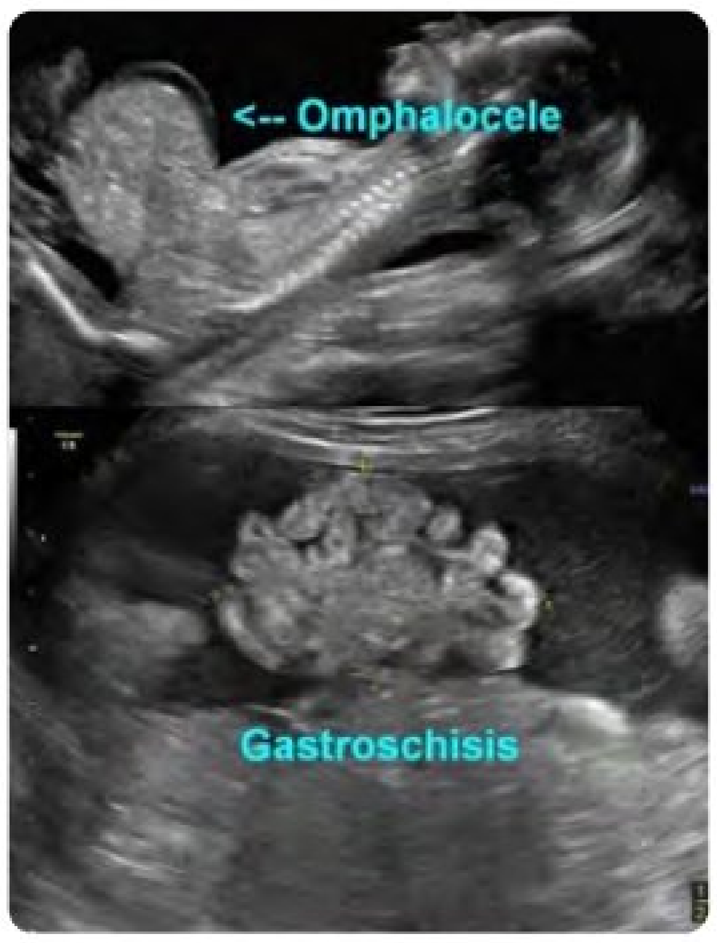
Gastroschisis → Defect is irregular contour



Ompalocele



Gastroschisis



7. USG also help in localising location of placenta, whether it is in upper segment (or) Lower segment

For localization of Placenta, USG should be done in 3rd trimester

Best time to detect Placenta previa on USG ⇒ 3rd Trimester

ANENCEPHALY

- Vault or skull is absent
- Adrenal glands are either absent or Hypoplastic

DHEA-S ↓ (or) Absent
↓
Decreased Estrogen Production by Placenta
↓
POST TERM PREGNANCY

- Polyhydramnios (d/t skull is absent, CSF leaks into amniotic fluids & also there is swallowing defects.)
↓
PRE TERM LABOUR

- MC Presentation is ⇒ Face presentation
- Chances of Recurrance ⇒ After 1 NTD : 4%
After 2 NTD : 10%

MC in →

FEMALE FETUS	MALE FETUS
Anencephaly	Macrosomia & Resp. defects

- Prevention of NTD ⇒ Folic acid Supplementation
- Management of Anencephaly ⇒ MTP

FOLIC ACID SUPPLEMENTATION

i. Prevents NTD
ii. Prevents Megaloblastic Anemia
iii. Prevent IUGR
iv. Prevents Abruptio placenta

DOSES OF FA DURING PREGNANCY

400mcg/day	1mg/day	4mg/day (Therapeutic dose)
All pregnant females NO with h/o NTD BABY (Prophyactic dose of FA)	If pregnant female has Megaloblastic Anemia	Pregnant female who has h/o previous NTD Baby
All pregnant female c̄ diabetes	If pregnant female has 2° Relatives c̄ h/o NTD Baby	Pregnant female is any antiepileptic drugs which ↑ chance of NTD Eg : Valproaic Acid
All pregnant females who all are on antiepileptic drugs which donot cause NTD Eg: LAMOTRIGINE	If pregnant female is on any drugs which ↓ absorption of FA Eg: Trimethoprim Sulfasalazine	Pregnant female who has sickle cell anemia (5mg > 4mg)

- Prophylactic dose of FA = 400 mcg
  ↓
  Prevent NTD
  ↓
  Ideally it should be given 3 months before pregnancy but atleast it should be given 1 month before pregnancy & it should be continued till 3 months after pregnancy
- Tablet of FA → Provided by Govt. of India
  ↓
  500mcg

- RDA : of FA in pregnancy → 500mcg
- If pregnant female has previous h/o NTD Baby

↓

4mg/day (Therapeutic dose)

↓

atleast it should be start from 1 month before pregnancy & continue it for 3 months after pregnancy

↓

After 3 months put her on ; 400mcg FA/day Throughout pregnancy

DIAGNOSIS OF ANENCEPHALY

Screening Test

↓

Maternal serum alpha fetoprotein (15-20 wks)

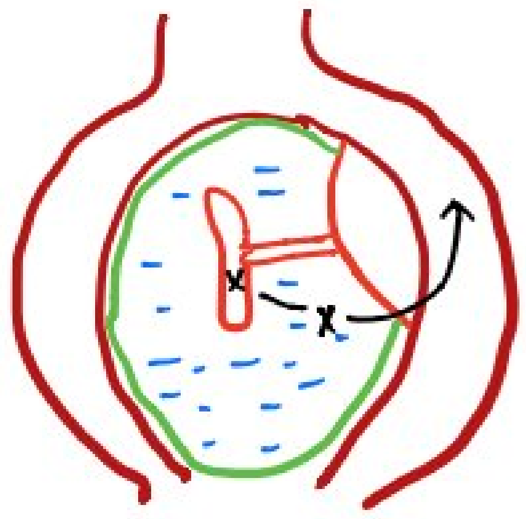
Best : 16-18 wks

Diagnostic Test

↓

Earlier : Amniocentesis (Invasive test)

Now : Level-2 scan/ TIFFA



Most sensitive biochemical marker for Anencephaly is ACETYL CHOLINESTERASE

ALFA FETO PROTEIN

- Synthesised in Early weeks → Yolk Sac
  Later on → Fetal GIT & Liver
- Major source : Fetal Liver
- Present in a) Fetal serum → Highest levels are @ 13 wks
  b) Amniotic fluids → Highest levels are @ 13 wks
  c) Maternal serum → Highest levels are @ 32 wks
- t½ : 5-7 day
- Screening test : Maternal serum alpha feto protein (≥ 2.5 MOM)
  In twin = ≥ 3.5 MOM

↑ AFP	↓ AFP
* NTD	* Diabetic : Diabetes
* Abdominal wall defects	* Gestational trophoblastic disease (Molar pregnancy, Choriocarcinoma)
* Pilonidal sinus	* O = Maternal obesity, overstimulated gestational age
	* Abortion
	* Trisomy Eg: Down syndrome

Q Which of the following is a best marker for gestational age ?

[✓ a) CRL at 16 wks]   c) FL at 32 wks
b) BPD at 16 wks   d) AC at 20 wks

(Because CRL at 16 wks is not measured)

Q Fetal sex can be detected by USG at

[✓ a) 14 weeks]   c) 18 weeks
b) 16 weeks   d) 20 weeks

Q Hormone Responsible for fetal growth

a) Growth Horm.   c) Parathyroid Hormone
[✓ b) Insulin]   d) Thyroxine

Q Fetal Kidneys start producing urine by

[✓ a) 3 months]   c) 5 months
b) 4 months   d) 6 months

Q Fetal stage starts at

JIPMER 2004

[✓ a) 9 wks]   c) 6 wks
b) 3 wks   d) 12 wks

Q Earliest cardiac activity can be detected by

[✓ a) 5 wks]   c) 7 wks
b) 6 wks   8) 8 wks

Since qn not saying TVS / TAS → Earliest it can be detected using TVS at 5 wks

Q Dose of FA in a pregnant female c̄ gestational diabetes

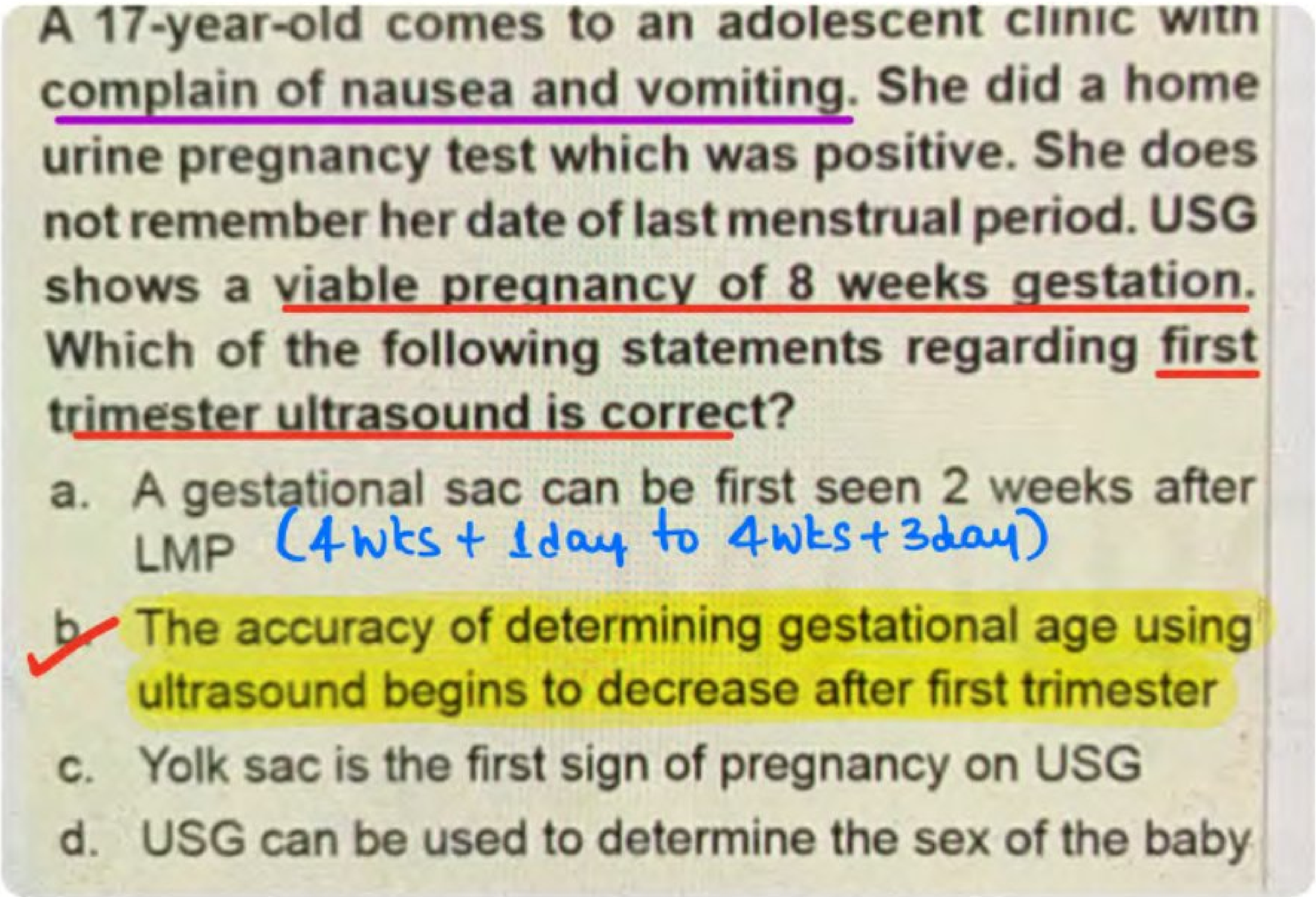
a) 4mg
[✓ c) 400 mcg]
b) 400mg
d) 40 mcg

Q Most sensitive biochemical marker for NTD

a) α-FP
c) pseudocholinesterase
b) acetylcholinesterase
d) PAPP A

Q All of the following are true about anencephaly except

a) More common in females
c) Folic acid supplementation preventive
b) ass with polyhydramnios
d) Enlarged adrenal gland



A 17-year-old comes to an adolescent clinic with complain of nausea and vomiting. She did a home urine pregnancy test which was positive. She does not remember her date of last menstrual period. USG shows a viable pregnancy of 8 weeks gestation. Which of the following statements regarding first trimester ultrasound is correct?

a. A gestational sac can be first seen 2 weeks after LMP (4wks + 1day to 4wks + 3day)

[✓ b. The accuracy of determining gestational age using ultrasound begins to decrease after first trimester]

c. Yolk sac is the first sign of pregnancy on USG

d. USG can be used to determine the sex of the baby

Q Increased α-FP is seen in

a) Down syndrome
c) Overstimulated gestational age
b) Molar pregnancy
[✓ d) Congenital Nephrotic Synd.]

PARTOGRAM

1. FETAL PART

: i) FHR - monitored every 30 min for 1 min

ii) Membrane : Intact +
Rupture - , if Ruptured - COLOUR should be mentioned

iii) Moulding : Over-riding of PARIETAL BONES of Baby

- 1+ - Bones touching each other
- 2+ - Overlaping but can be separated.
- 3+ - Overlaping and cannot be separated.

Overlaping of Bones occurs when space in PELVIS is less.

2. PROGRESS OF ACTIVE PHASE

: Alert line $\xrightarrow{4hrs}$ Action line

If pt comes ≤ 6cm dilatation ⟶ Plotting begins on ALERT LINE

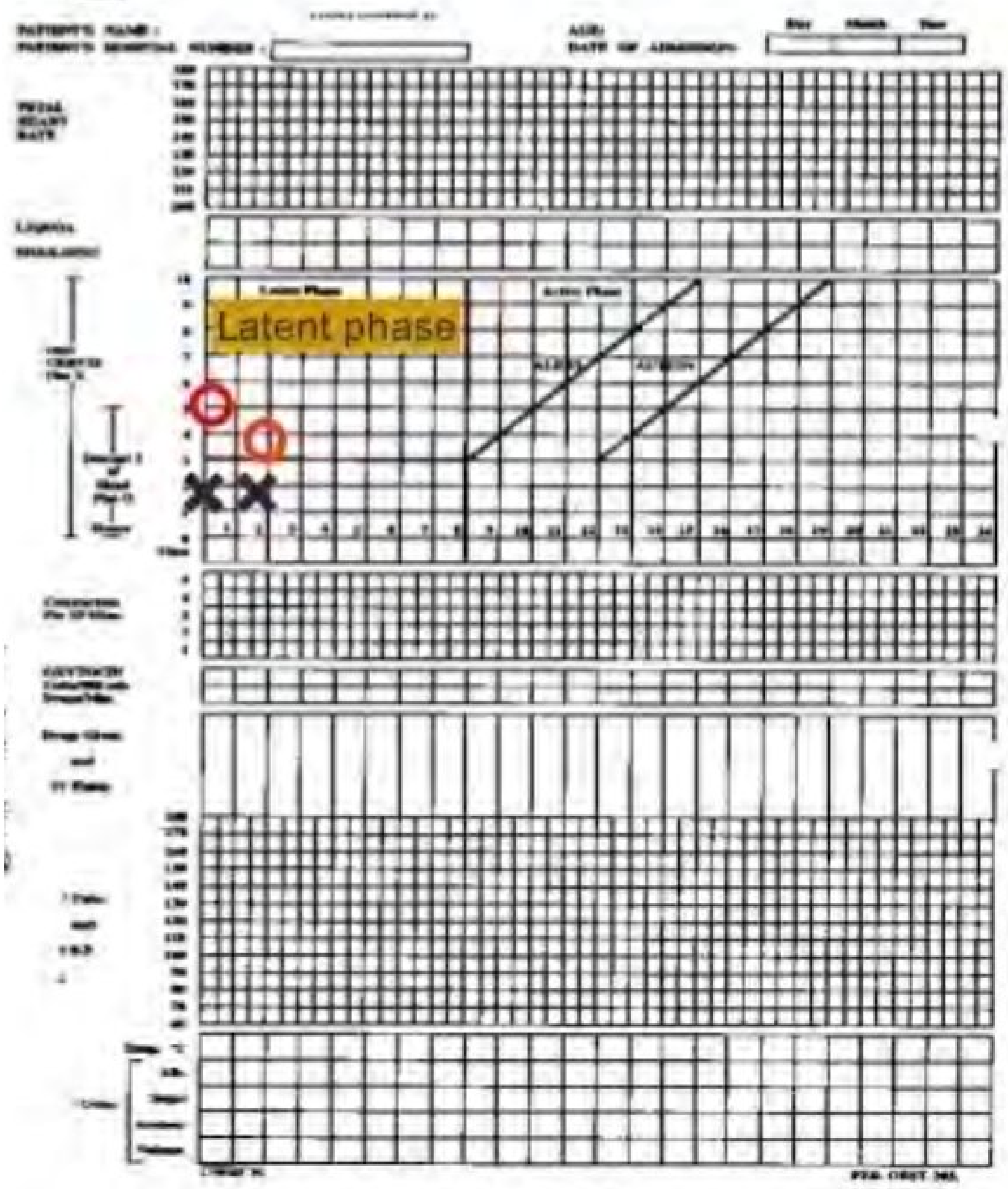
- If any plotting : is on Ⓛ/on ALERT LINE - Ⓝ
  - is on Ⓡ of ALERT LINE → SLOW PROGRESS ⟶ Referred to HIGHER CENTRE
  - lies on ACTION LINE or to Ⓡ of ACTION LINE → INTERVENTION needed.

P/v examination done 4hrly.

3. MATERNAL PART

- Contractions - every 30mins for 10 min. Check for how many contractions.
- Monitor Vitals - PR/ RR / Temp / u/o / urine Albumin
- Contraction represented
  - ░ - last < 20sec
  - ▨ - 20-40 sec
  - ■ - > 40 sec

PARTOGRAM RECORDING



Labour progress recording in latent phase

Plot dilatation as "**X**"
Plot descent as "**O**"

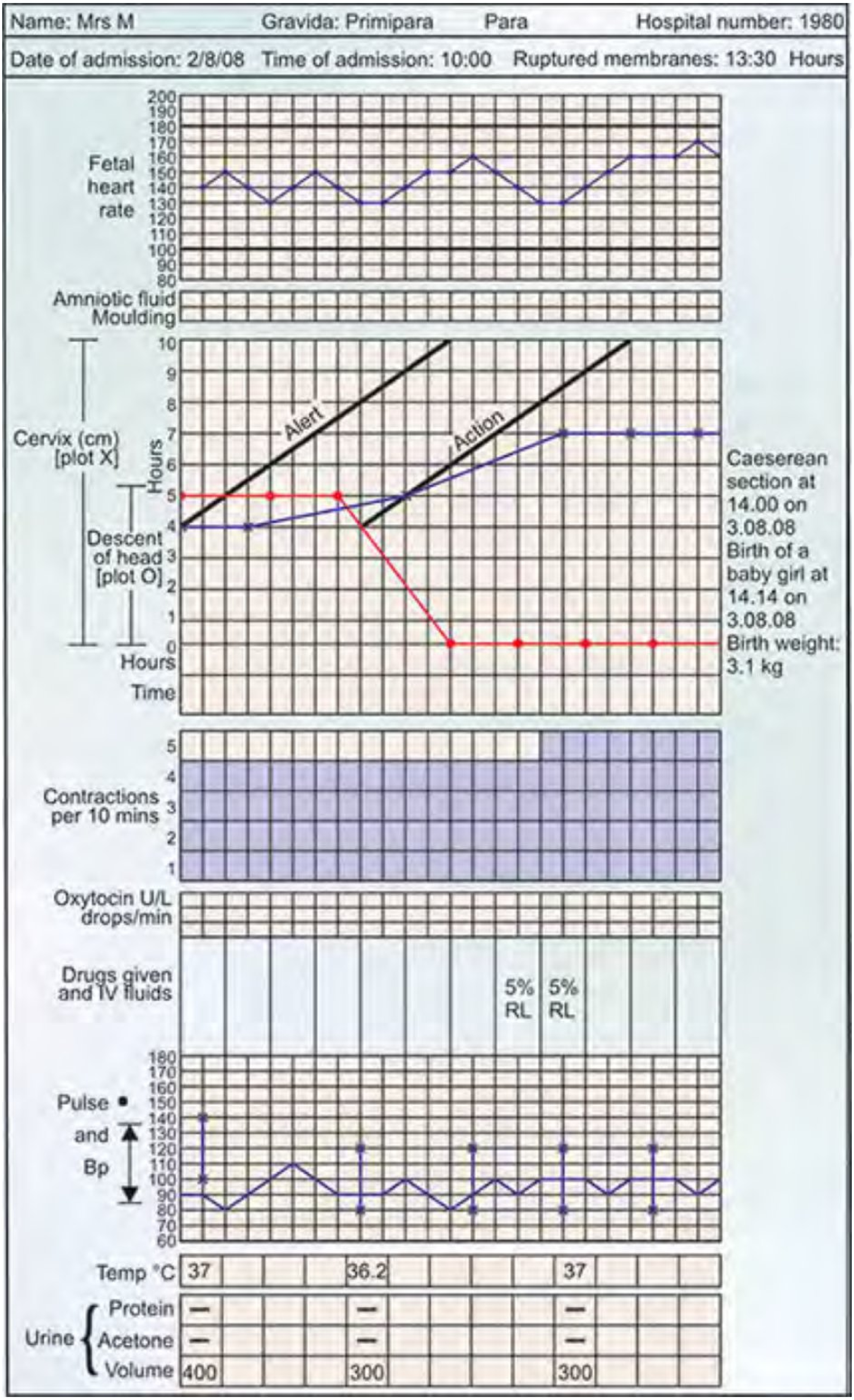
At admission:

- Dilatation → 2 cm
- Descent → -2

2 hours after admission:

- Dilatation → 2 cm
- Descent → -1

As the dilatation is only 2 cm therefore the labour progress is in the latent phase


Name: Mrs M Gravida: Primipara Para Hospital number: 1980

Date of admission: 2/8/08 Time of admission: 10:00 Ruptured membranes: 13:30 Hours

Caeserean section at 14.00 on 3.08.08

Birth of a baby girl at 14.14 on 3.08.08

Birth weight: 3.1 kg

Drugs given and IV fluids: 5% RL 5% RL

Temp °C: 37 36.2 37

Urine Protein: — — —

Urine Acetone: — — —

Urine Volume: 400 300 300

LIE

- Relation b/w long axis of fetus & long axis of uterus

- Correct DEXTRO ROTATION - 1st

i) Longitudinal ii) Transverse

PRESENTATION

- part of fetus which is foremost to birth canal

LEOPOLD MANEUVERS

1st GRIP : FUNDAL GRIP - Broad Soft Part
S/o Buttocks

2nd GRIP : LATERAL GRIP

- Smooth curved part → S/o BACK
- Small knobby irregular → S/o LIMBS

3rd GRIP : PAWLIK'S GRIP

- Hard, Round, Smooth Structure - S/o HEAD
- if can be moved side to side - FREE FLOATING or BALLOTABLE

4th GRIP : DEEP PELVIC GRIP

- Both Hands
  - CONVERGING : head has not entered pelvis
  - DIVERGING : head has entered pelvis

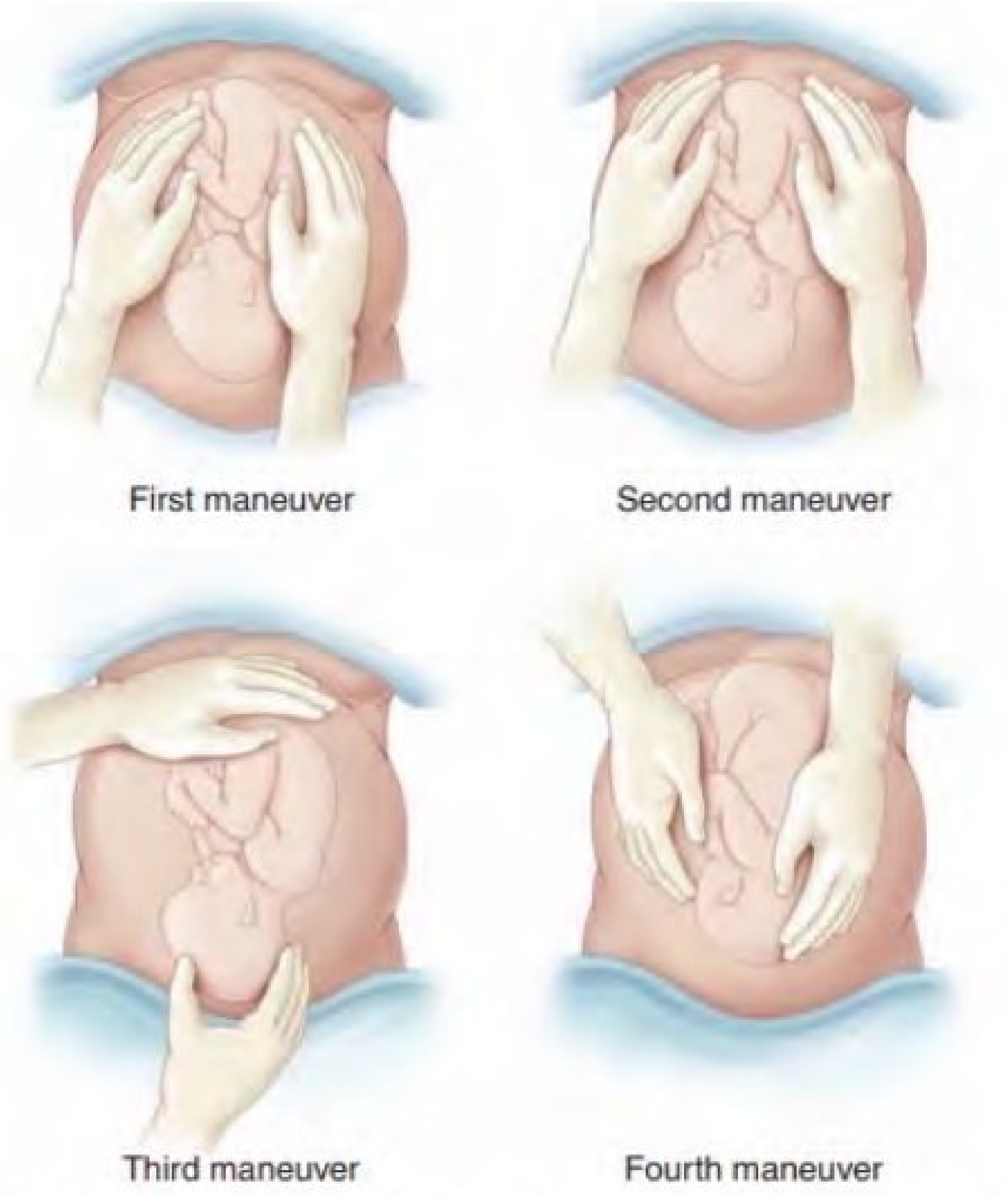


FIGURE 9.7. Leopold maneuvers. The maneuvers are used to determine fetal position: 1) determination of what is in the fundus; 2) evaluation of the fetal back and extremities; 3) palpation of the presenting part above the symphysis; 4) determination of the direction and degree of flexion of the head.

- PRESENTATION can be **① CEPHALIC** **② BREECH** **③ SHOULDER**

ATTITUDE : Flexed/Extended Head

1. VERTEX → Well flexed → Ⓝ Labour
→ Deflexed

2. PARTIAL EXTENSION → aka BROW PRESENTATION

3. FULL EXTENSION → FACE PRESENTATION

ANTERIOR FONTANELLE or BREGMA } DIAMOND / RHOMBOID

POSTERIOR FONTANELLE or LAMBDA } TRIANGULAR

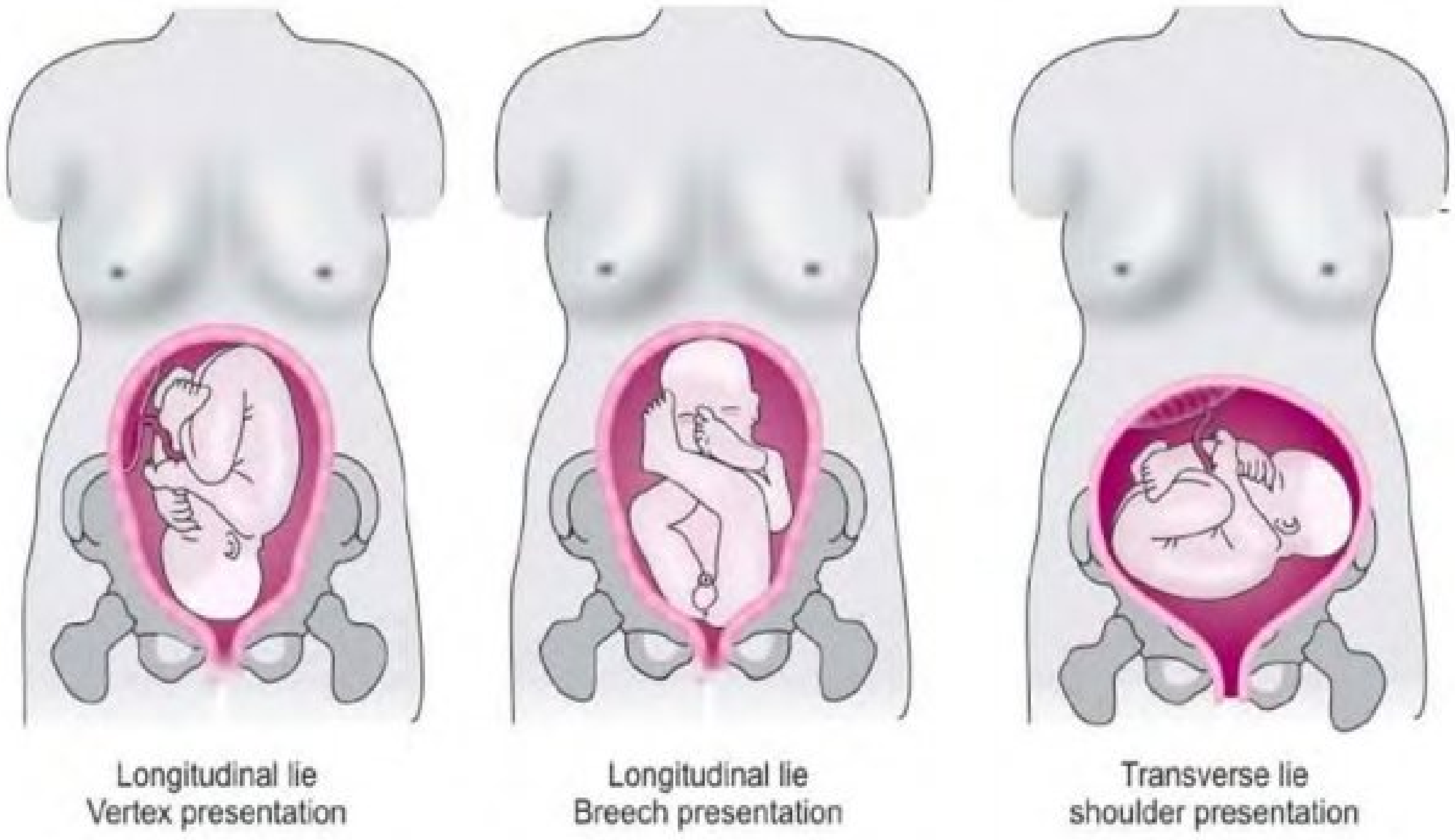
VERTEX - BOUNDARIES
→ ANTERIOR → ANTERIOR FONTANELLE + CORONAL SUTURE
→ POSTERIOR → POSTERIOR FONTANELLE + LAMBDOID SUTURE

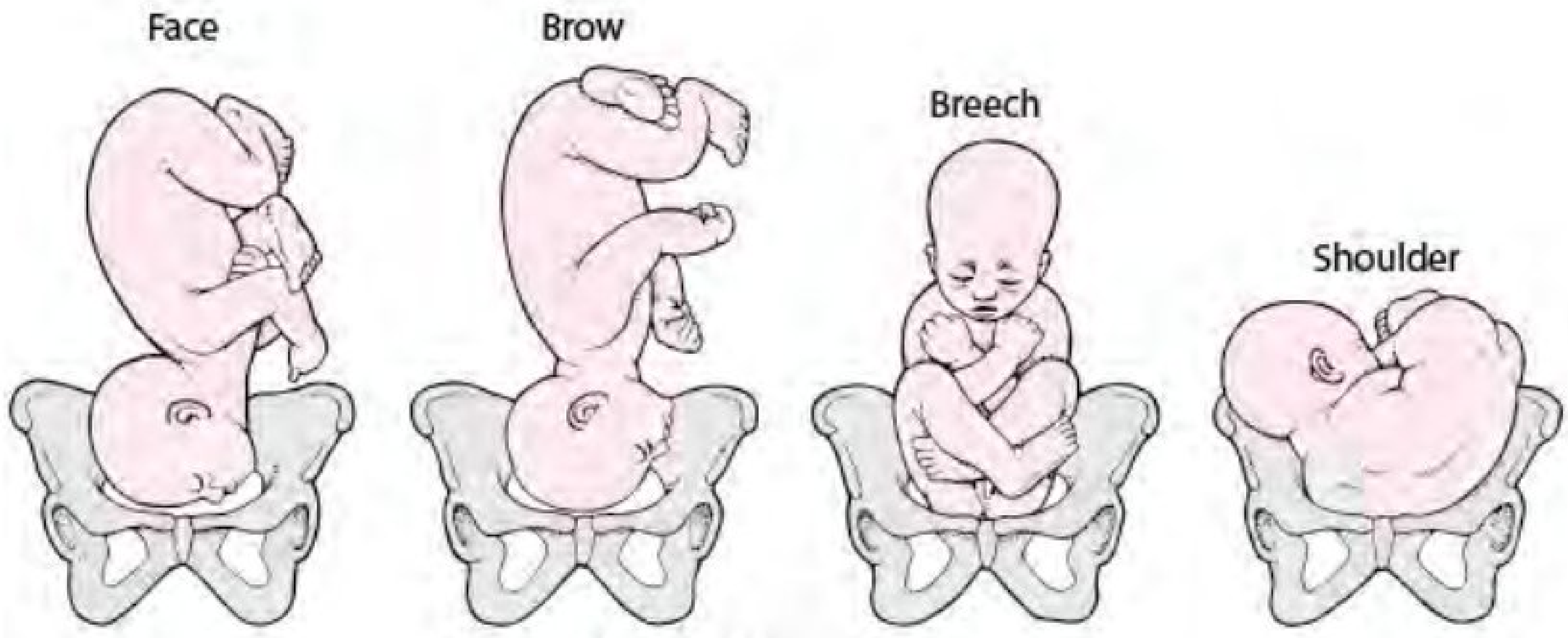
BROW - BOUNDARIES
→ ANTERIOR → GLABELLA + SUPRAORBITAL RIDGE
→ POSTERIOR → ANTERIOR FONTANELLE + CORONAL SUTURE

FACE - More Anterior to BROW

On WELL FLEXED HEAD } SINCIPUT at HIGHER level than OCCIPUT
→ On P/v - Posterior Fontanelle/Occiput
→ On P/A - SINCIPUT

DEFLEXED HEAD : SINCIPUT at SAME level as OCCIPUT





Abnormal Presentations

ENGAGEMENT

- when the largest fetal head diameter crosses the pelvic brim.

[Bi Parietal Diameter]

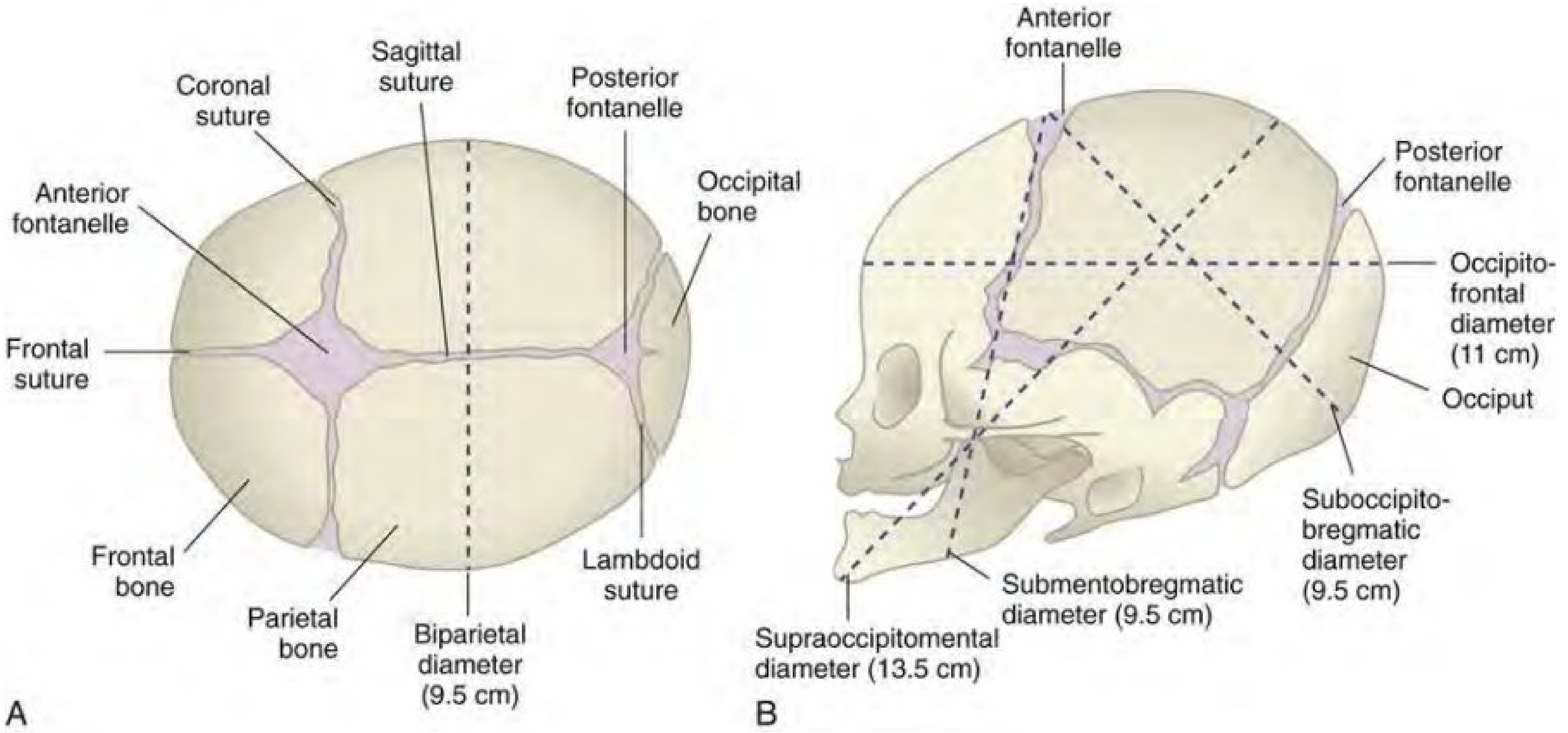
- Largest Transverse Fetal Head diameter in Well flexed head (VERTEX) – BPD - 9.5cm

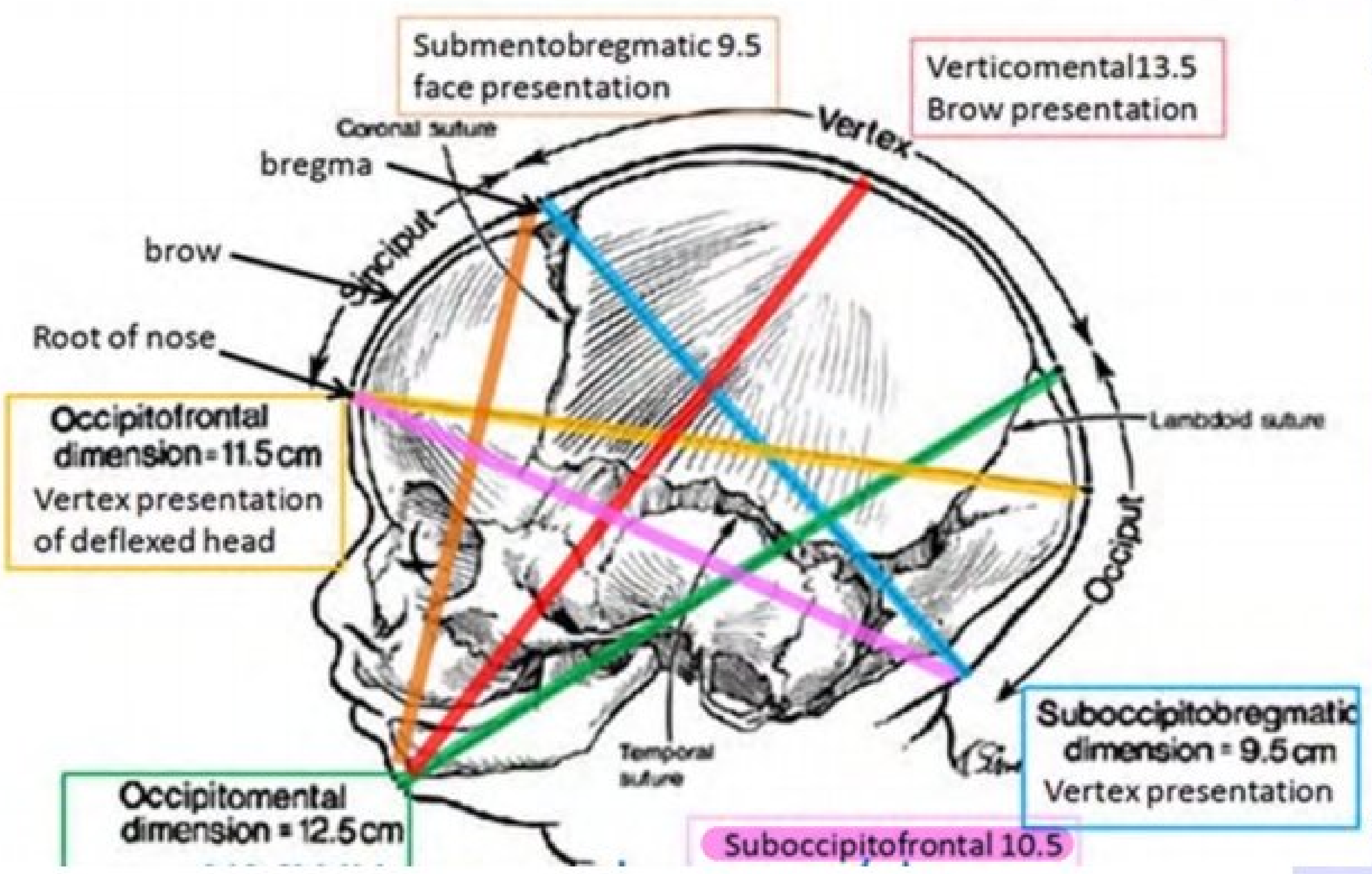
AP Engaging diameter change ≤ presentation [Degree of Flexion/Extension]

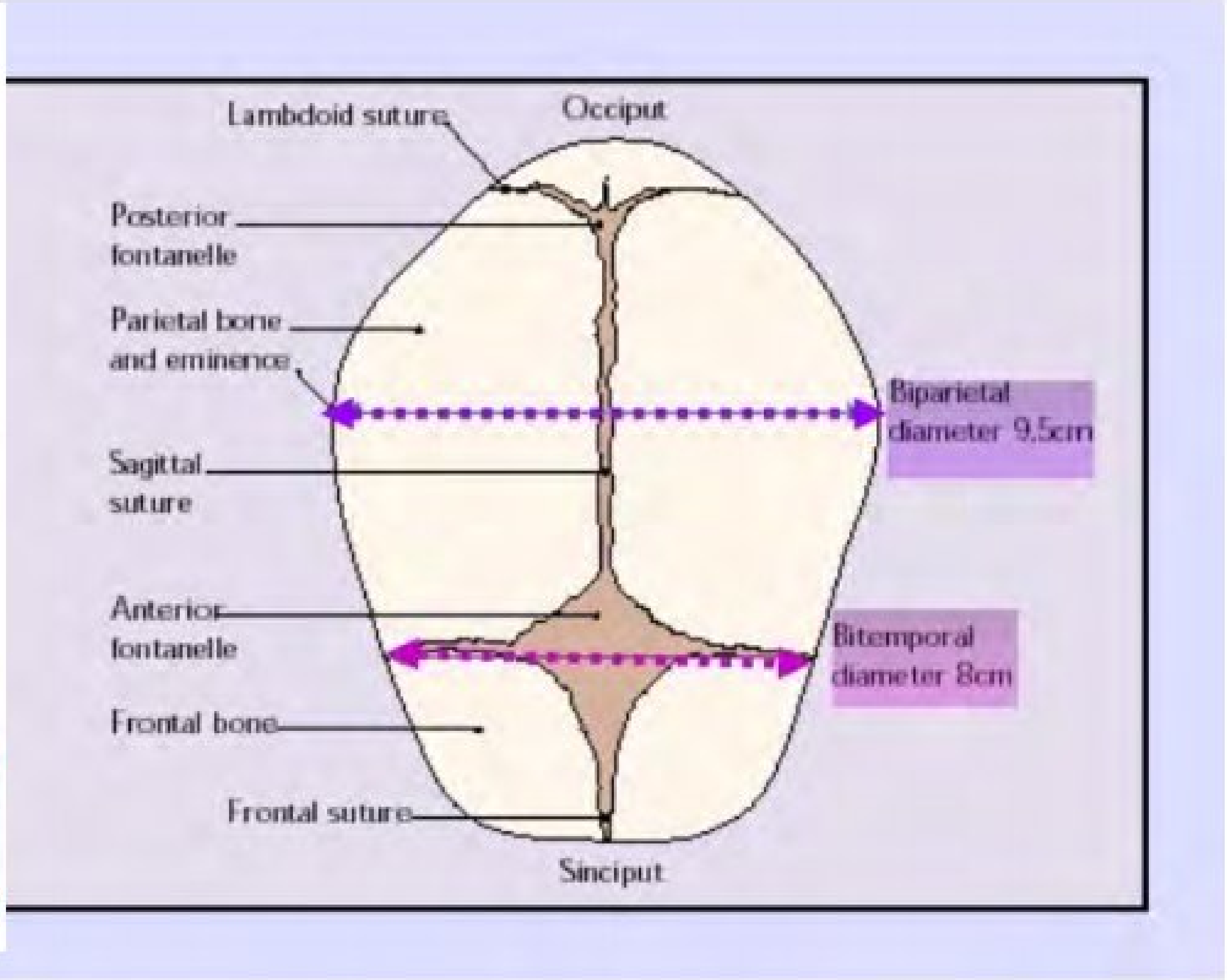
1. Well Flexed / VERTEX - Suboccipito Bregmatic → centre of AF - 9.5cm
2. Deflexed [Engagement Delayed] → Occipito frontal → Ant. end of Bregma - 11.5cm
   → Suboccipito frontal - 10.5cm
3. Brow - Mento-vertical - 14cm [Largest Fetal Head Diameter]
   ↳ Highest point of Sagittal Suture

SMALLEST Head diameter - BIMASTOID (7.5cm) > BITEMPORAL (8cm)

4. Face - Submentobregmatic (9.5cm)







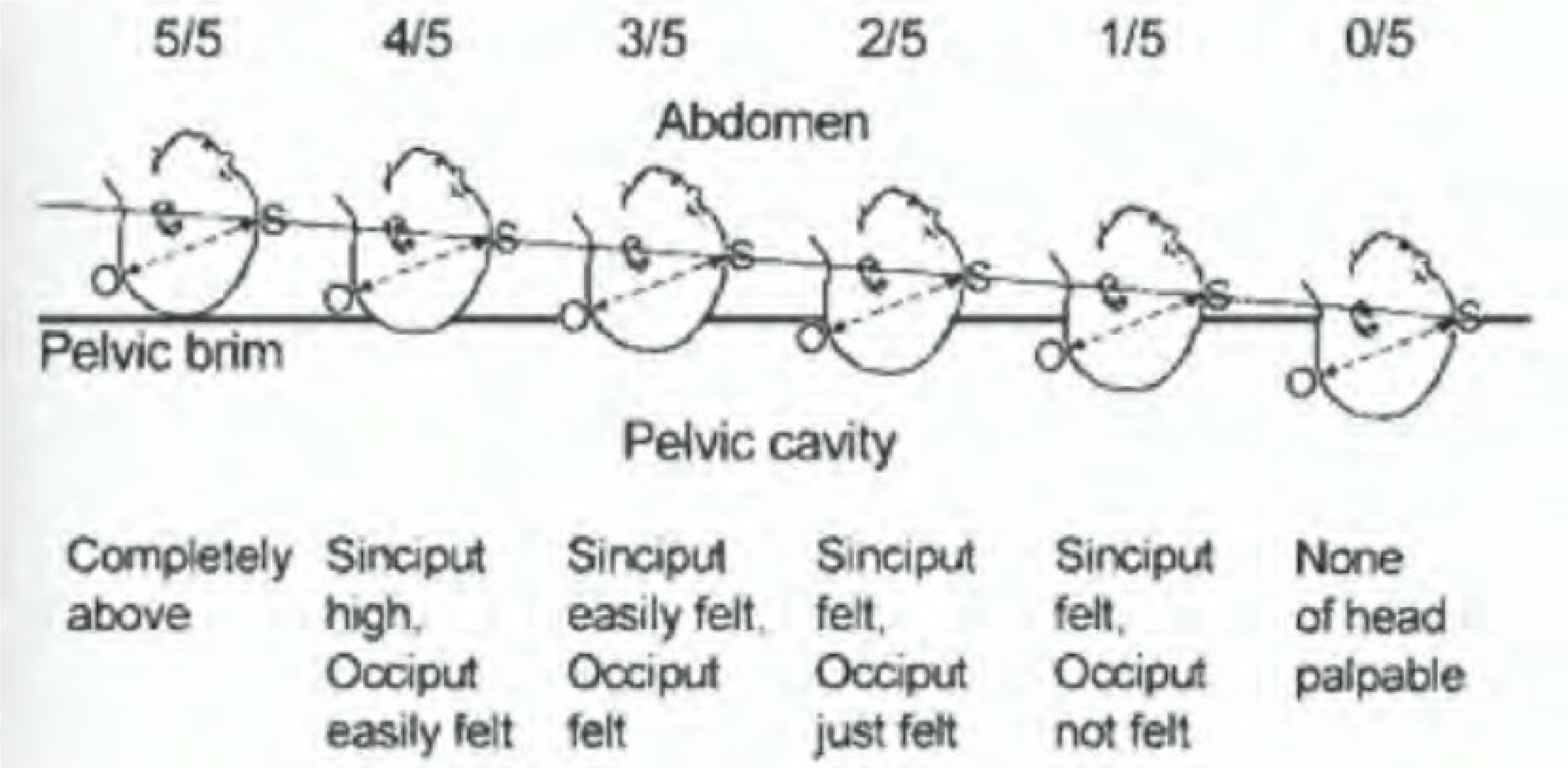
Engagement rules out CPD at Pelvic brim

On P/A : Rule of Fifths ⟶ Fetal Head into 5 equal parts. Eg: 5/5, 4/5, 3/5, 2/5, 1/5, 0/5

$$\frac{\text{Numerator}}{\text{Denomenator}} = \frac{\text{How many parts above brim}}{\text{Total Equal parts}}$$

5/5 - Free Floating ; 4/5 - Fixed head

2/5 - Engaged.


5/5
4/5
3/5
2/5
1/5
0/5
Abdomen
Pelvic brim
Pelvic cavity
Completely above
Sinciput high, Occiput easily felt
Sinciput easily felt, Occiput felt
Sinciput felt, Occiput just felt
Sinciput felt, Occiput not felt
None of head palpable

On P/v : Leading point of head c̄ respect to ISCHIAL SPINE → STATION 'O' ; '-5' to 'O' to +5

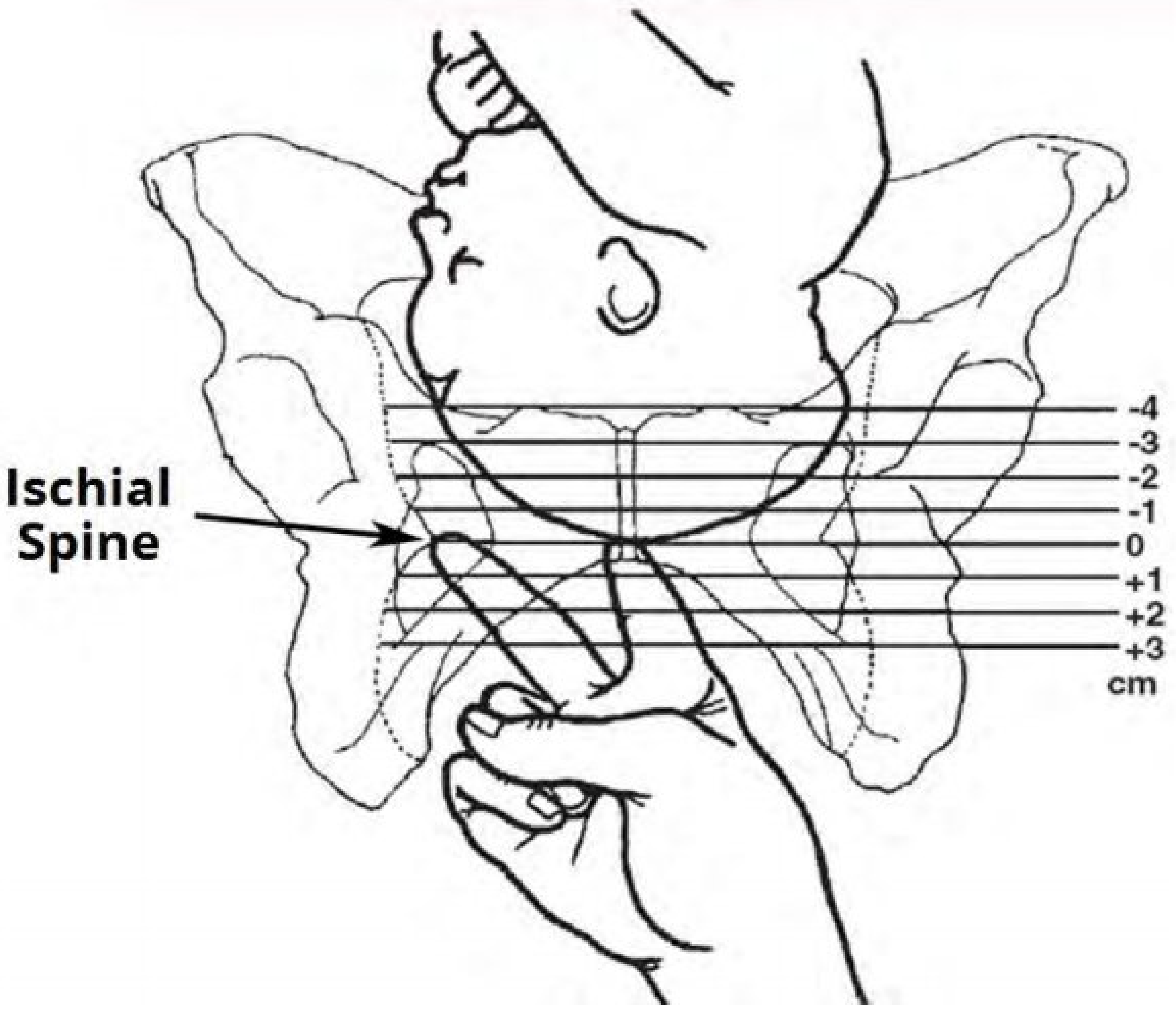
at **STATION 'O'** - BPD crossed into Pelvic brim; Engaged head - 2/5

at **STATION '-2'** - Leading point is 2cm above ischial spine

In Primi - engagement happens at 37wks

In Multi - engagement happens at ONSET OF LABOUR

Primi - free floating head at 37wks
cause : 1. Deflexed head - Ⓜ︎Ⓒ
2. CPD
3. Placenta Praevia at OS
4. Polyhydraminos



POSITION : Location of head in pelvis

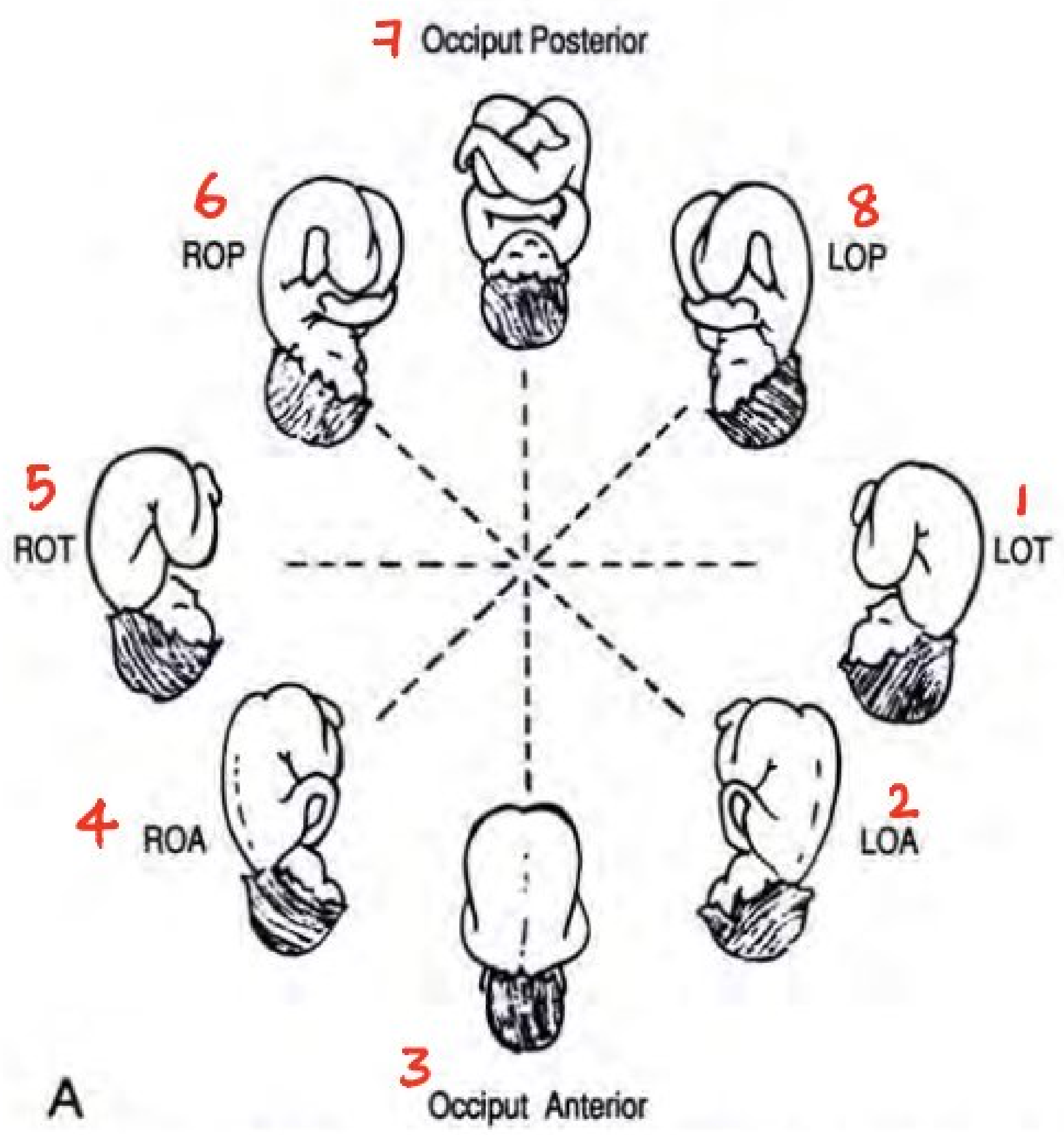
VERTEX	BREECH	FACE	BROW
↓	↓	↓	↓
OCCIPUT (O)	SACRUM (S)	MENTUM (M)	FRONTAL BONES (F)

2,3,4 ⟶ Bony prominences close to Pubic Symphysis - Ant
6,7,8 ⟶ Bony prominences close to Sacrum - Post
1,5 ⟶ TRANSVERSE

L - Left
R - Right } Side of Mother

(MC) Position of Baby in Labour - LOT

STEPS OF NORMAL LABOUR



1. ENGAGEMENT - 2/5

2. DESCENT } of HEAD
3. FLEXION }

4. INTERNAL ROTATION Ⓘ(IR) OF

i) HEAD happens by 2/8

ii) POSTERIOR SHOULDER by 1/8 } Same direction as
iii) ANTERIOR SHOULDER by 1/8 } Head

after IR Occiput lie - under the pubic symphysis

after IR Shoulder lie - Ⓛ oblique
↳ Ⓛ Sacro iliac joint of Mother

5. DELIVERY OF HEAD by EXTENSION

6. EXTERNAL ROTATION : 1st 1/8th - only HEAD moves - aka RESTITUTION
2nd 1/8th - Both Head and Shoulder moves

Rotation direction of movement : IR and ER - Opposite
ER and Restitution - Same

7. DELIVERY OF SHOULDER and REST of Body by LATERAL FLEXION

CROWNING : when BPD stretches the vulval outlet and doesnot RECEDE back in b/w contractions

• Perineum is Stretched and EPISIOTOMY can be given.

What makes LABOUR AbⓃ ? 3 P's

- I. PELVIS
- II. PUSH
- III. PASSENGER or Fetus
  - VERTEX
    - CPD
    - Occipito posterior
  - NON-VERTEX

CLASSIFICATION OF PELVIS - CALDWELL and MALLOY

1. GYNECOID PELVIS - (MC), Inlet - almost circular, s/i 50%

2. ANTHROPOID PELVIS - 2nd (MC), Inlet - AP Oval, s/i 25% of Females DEEPEST PELVIS

3. ANDROID PELVIS - Typical of Male, Inlet - Heart shaped, s/i 20% of females

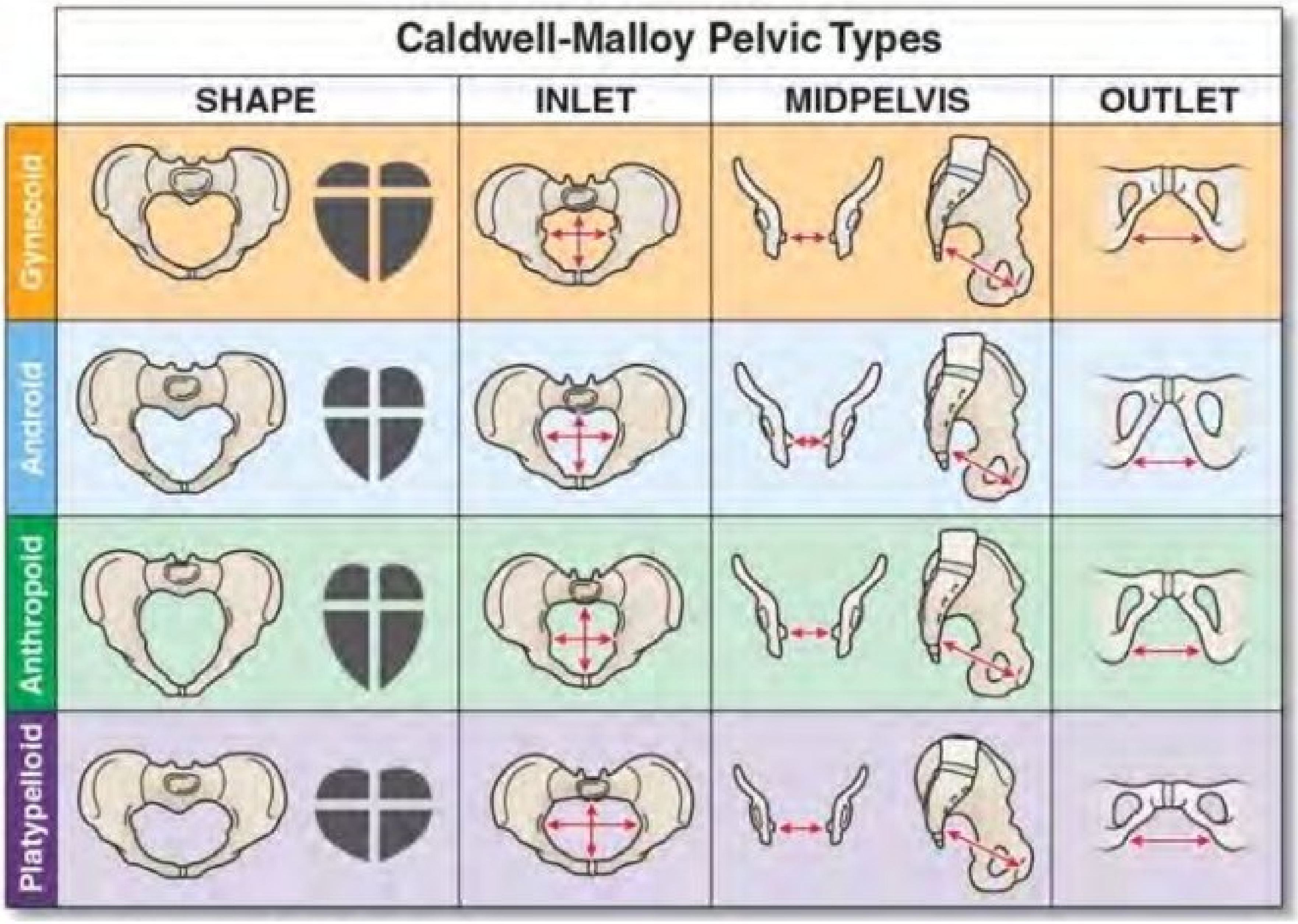
4. PLATYPELLOID aka FLAT GYNAECOID - Inlet - Transverse Oval, s/i 5% females

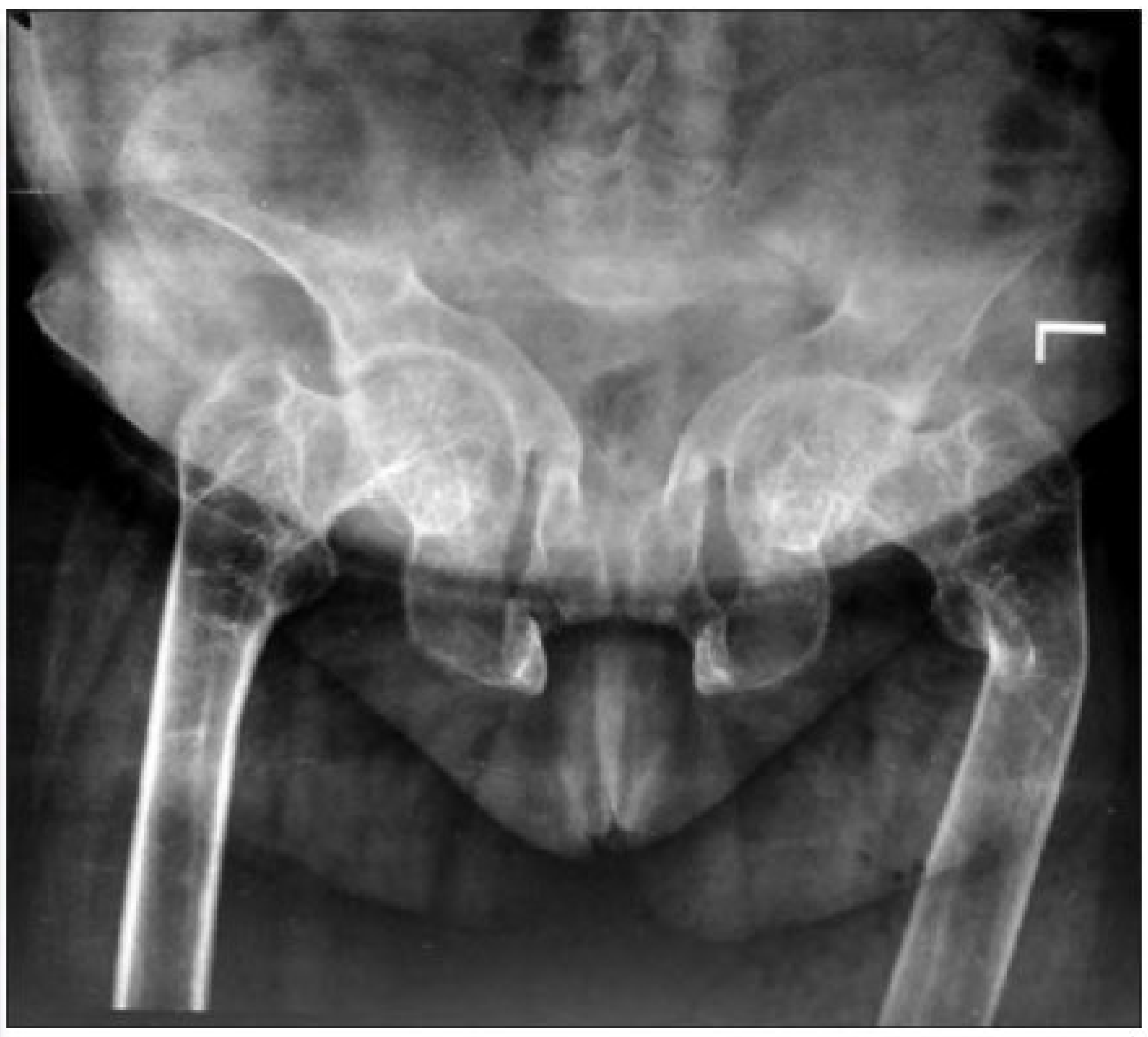
5. NAEGELES PELVIS - one ALA of Sacrum absent

6. ROBERT'S PELVIS - BOTH ALA of Sacrum absent

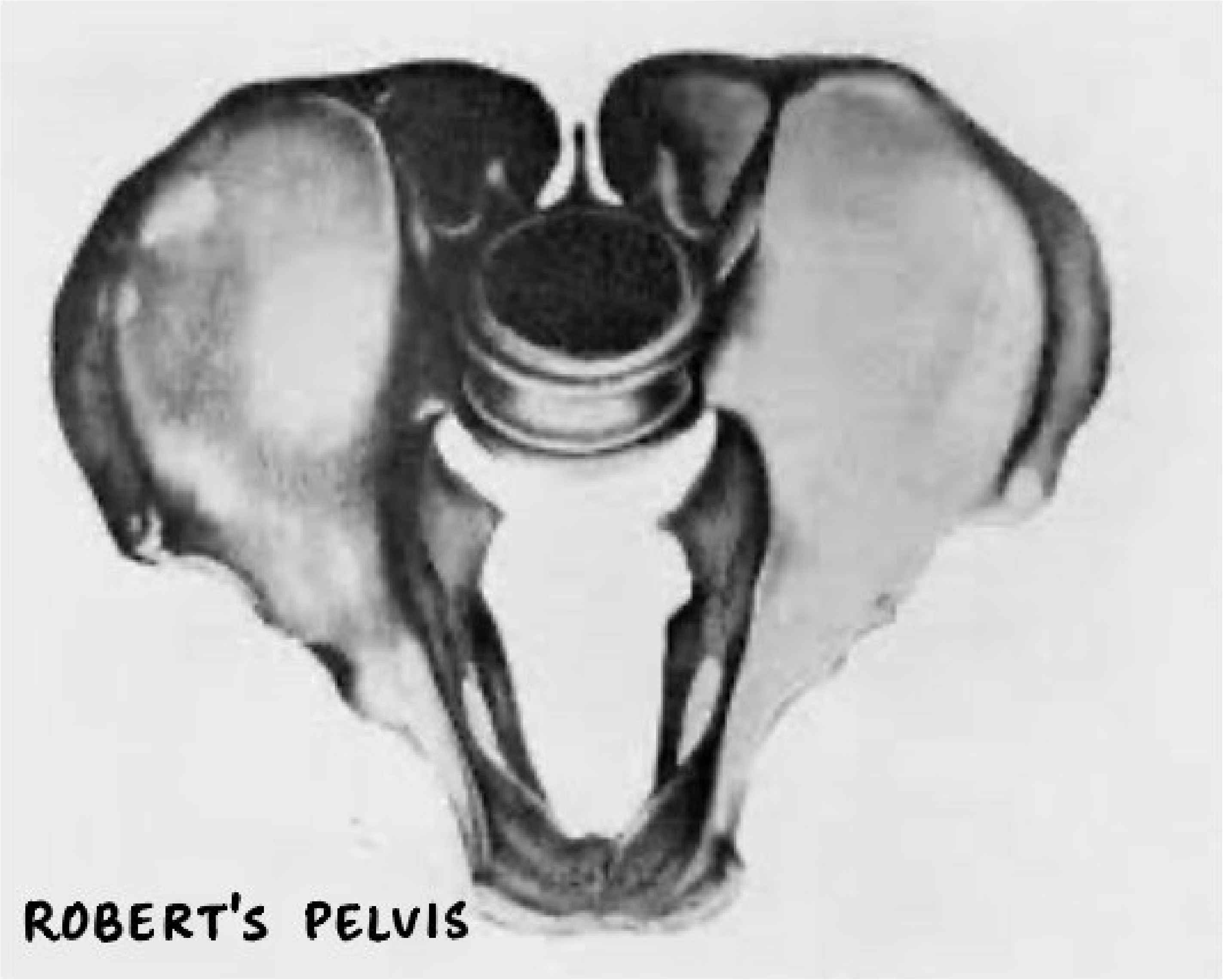
} deformed Pelvis → Ⓝ vaginal delivery NOT possible
↓
So, C.S done

7. TRI RADIATE PELVIS - s/i Vit. D deficiency, Osteomalacia, Rickets

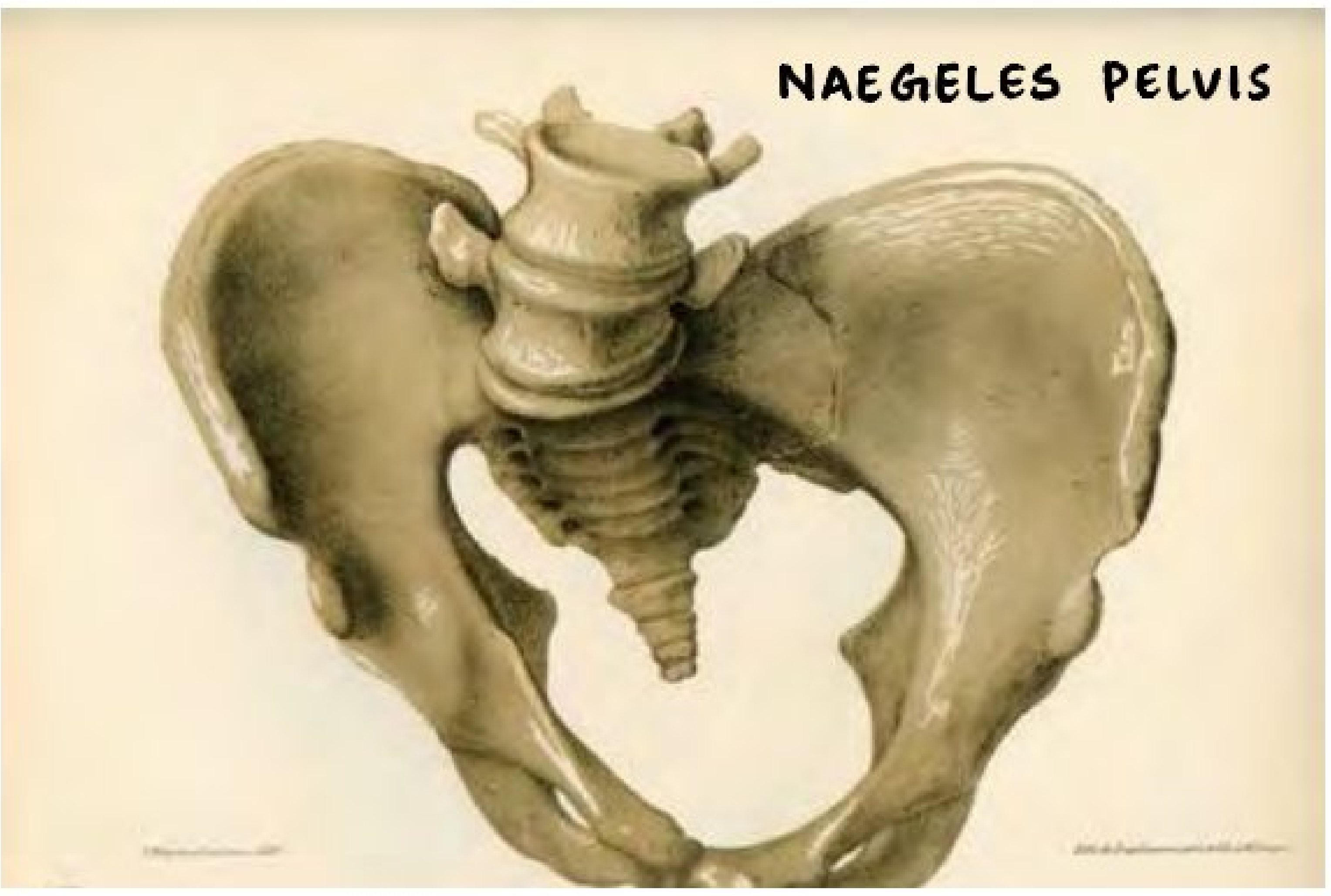




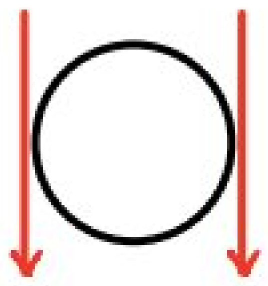
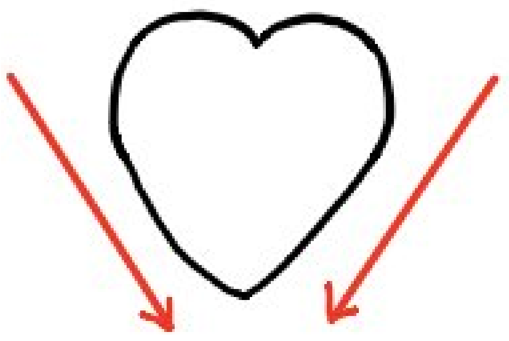
X-Ray showing Tri radiate pelvis
Separation of pubic symphysis and
COXA VARA deformity of neck of
Ⓛ femur



ROBERT'S PELVIS



NAEGELES PELVIS

GYNECOID	ANDROID
 Side walls PARALLEL Ischial spines - Blunt Subpubic angle - OBTUSE (90-100°) Shallow Pelvis	 Side walls converging Occiput goes posterior (MC) cause of OCCIPITO POSTERIOR Ischial spines - Sharp Head can get stuck → Deep Transverse Arrest Subpubic angle 85° Deep Pelvis

I. PELVIS

INLET Shortest : AP diameter → 3 in Number

1. Diagonal conjugate
2. Obstetric conjugate - Ⓝ 10.5cm — {Most important but it cannot be assesed clinically
3. True Conjugate

- Only Diagonal conjugate can be assesed

Ⓝ Fingers not touch Sacral promontory, then DC = 12

DC - 1.5 = OC

CAVITY Shortest : Transverse — Inter ischial Spine Diameter ; Both spine cannot be touched together
Ⓝ 10.5cm

OUTLET Shortest : Transverse — Intertuberous Diameter (ITD) ; FOUR KNUCKLE Test
Ⓝ 11cm
Ⓝ easily fit b/w Ischial tuberosity = 11cm

CONTRACTED PELVIS

- small pelvis even for averaged size baby
- one or more of these diameter are small
- Absolute Indication for C.S
- Pelvic Assessment is done in Primi b/w 38-39 wks

① Inlet : If OC < 10cm ; DC < 11.5cm

② Cavity : if IID < 8cm (Diagnostic)
< 10cm S/o Contracted cavity

③ Outlet : if ITD < 8cm

CEPHALO PELVIC DISPROPORTION [C.P.D]

- pelvis not grossly small
- TRIAL OF LABOUR - Best way to look for CPD → After delivery - MRI, CT, X-Ray Perimetry } done for Pelvic assessment

End point of Trial of Labour for C.P.D

Deliver
Arrest
Fetal distress → C.S

CPD → slow progress despite uterine contractions → Arrest → C.S done
↓
If C.S not done
OBSTRUCTED LABOUR

OBSTRUCTED LABOUR

- On General Physical Examination
  - Exhausted
  - Dehydrated
  - Tachycardia
  - Tachypnea

On P/v
1. Caput
2. Moulding
3. Hot Dry Vagina

if empty the bladder → HEMATURIA

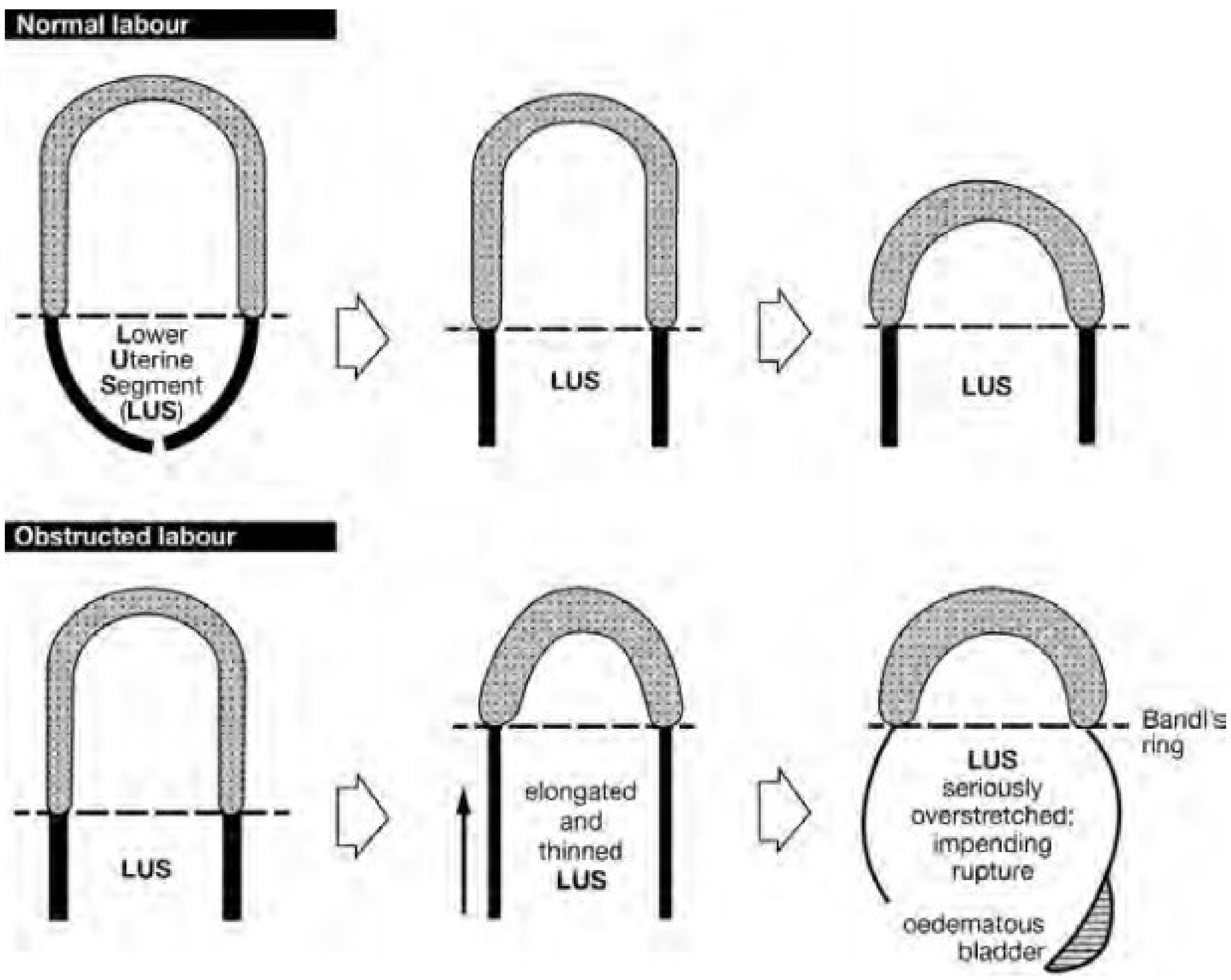
Mx: C.S even if FHR is ABSENT

- Previously if FHR is ABSENT destructive procedure were carried out:
  1. CRANIOTOMY
  2. SYMPHYSIOTOMY

POST C.S

Ischemia → Slowly Shed off → Leakage of Urine
↓
Vesico vaginal Fistula

On P/A
- upper segment - Tonically contracted
- Lower segment - Thinned and stretched
- B/w 2 segments - Ring (depression) aka BANDL's Ring - Pathological Retraction Ring
- FHR - severe distress or absent d/t suprapubic bulge full bladder


Normal labour
Lower Uterine Segment (LUS)
LUS
LUS
Obstructed labour
LUS
elongated and thinned LUS
Bandl's ring
LUS seriously overstretched; impending rupture
oedematous bladder

(MC) cause VVF in developing countries - OBSTRUCTED LABOUR

Q. $G_2P_1L_1$ c Prev C.S for CPD → Vaginal delivery can be done if no CPD in Current Pregnancy.

II. PUSH

- Uterine contractions → ↑ Intra Uterine Pressure - at 10 mm Hg - Uterus hard (Palpable)
  - at 15 mm Hg - painful - Initiate Cervical dilatation
- Adequate Contractions: generate 220 - 250 (>200)

MONTE VIDEO UNITS (units of uterine contraction)
= No. of contractions in 10 min × Pressure generated by each. Eg: 3 × 40 mmHg = 120

- If Adequate contractions ⟶ No further Augmentation required
- Touch FUNDUS c̄ INDEX Finger → if cannot be indented ⟶ 40mm Hg ⟶ MODERATE Strength Contractions

1^{st} Stage - at end - 50mm Hg

2^{nd} Stage - at end - 80mm Hg

- **PACEMAKER of Uterine Contraction - Ⓡ cornua**

Contractions are strongest at FUNDUS. **HYPERSTIMULATION** ⟶ Tachysystole c̄ Fetal distress

1. Stop OXYTOCIN drip
2. Ⓛ Lateral position
3. Administer O_2 by mask
4. Administer i.v fluids Fast

FHR will become Ⓝ ⟵ We can Restart Oxytocin after 30 min

Restart at HALF Previous dose ⟶ if contractions are INADEQUATE and LABOUR slow → Do AUGMENTATION = ARM + OXYTOCIN

↓

if ADEQUATE

→ CPD → Mx. TRIAL of LABOUR → Deliver / Arrest

→ it can be Occipito Posterior Position

Ⅲ. PASSENGER

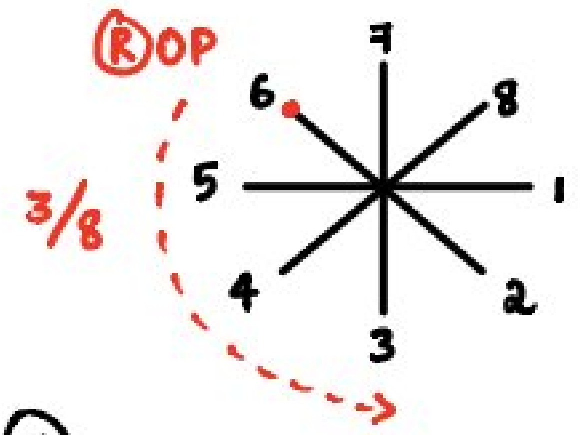
NON VERTEX
- Malpresentations

VERTEX

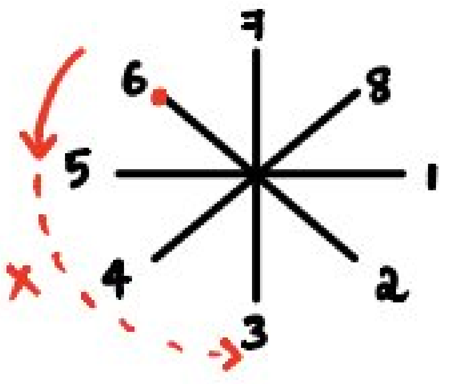
if AbⓃ Labour; CPD; Occipito posterior ⟶ Malposition

- (MC) cause of Occipito posterior - ANDROID PELVIS
- 2^{nd} (MC) cause → DEFLEXED Head ⟶ Engaging diameter is Occipito frontal or Suboccipitofrontal
- (MC) position in OP ⟶ ⓇOP ⟶ Outcomes in ⓇOP → 3 P's are Ⓝ, slow Labour despite adequate contraction
  Mx: Wait and Watch

In Ⓝ Labour - 2/8



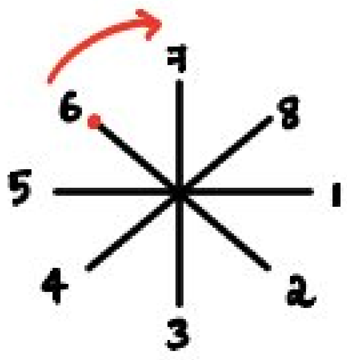
- 2^{nd} Outcome : if Pelvis AbⓃ
  eg: ANDROID Pelvis
  - Deep Transverse Arrest



FACE Presentation s/i
PLATYPELLOID Pelvis

3rd Outcome : In ANTHROPOID Pelvis - Direct OP

Vaginal delivery possible
Face to Pubis → C.S ← Persistent OP



INDUCTION OF LABOUR

Contraindicated in wherever V.D is not done

1. Contracted Pelvis
2. Placenta praevia
3. Transverse lie (Malpresentations)
4. Prev. h/o Rupture uterus
5. Classical C.S
6. ≥ 3 LSCS

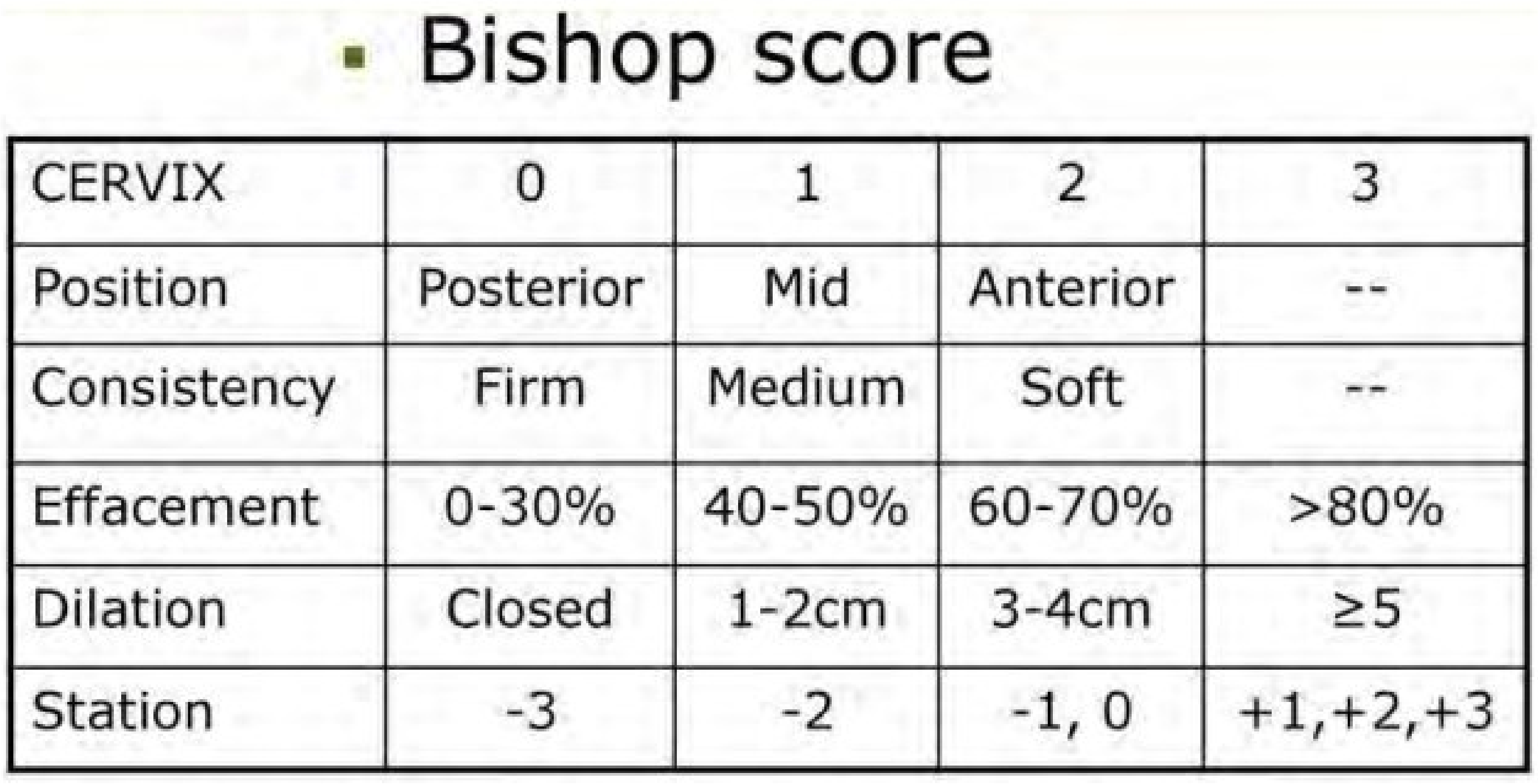
Pre-Induction Scoring [BISHOP's SCORE]

- most impt - Dilatation
  least impt - Position

- In Modified Bishops →
  Effacement is replaced by cervix length

- In Simplified Bishops →

  Dilatation, Effacement, Station



Bishop score

CERVIX	0	1	2	3
Position	Posterior	Mid	Anterior	--
Consistency	Firm	Medium	Soft	--
Effacement	0-30%	40-50%	60-70%	>80%
Dilation	Closed	1-2cm	3-4cm	≥5
Station	-3	-2	-1, 0	+1,+2,+3

- In BISHOP's Score ≥9/13 → FAVOURABLE → on IOL → LABOUR - Successfull
  ↳ ≤5 (<6) → UNFAVOURABLE → Do cervical Ripening → INDUCE CONTRACTION

CERVICAL RIPENING

Medical
- done (MC)

Mechanical
1. Osmotic devices - LAMINARIA TENTS
2. BULB of FOLEY's Catheter

DRUGS

1. PG E_2

- CERVIPRIME
- DINOPROSTONE (gel)

0.5mg →

- Intracervical but can be put in the post. fornix
- Repeat 6hrly
- Maximum 3 Application

Then → Start OXYTOCIN after 6hrs

Third Stage of Labor & Atonic PPH

THIRD STAGE OF LABOR

- Begins after delivery of baby & ends at the delivery of Placenta

↓

Plays crucial Role in PPH

↓

if 3rd stage managed properly ⇒ PPH Can be prevented

- Average duration of 3rd Stage ⇒ 15-20 min
- If 3rd stage is ≥30 min ⇒ Prolonged third Labour / Retained Placenta
- 3rd stage can be maintained by

PASSIVELY

- Duration : 15-20 min
- Waiting for spontaneous expulsion of Placenta
- Chances of PPH & maternal mortality are more.
- Since human, placenta is deciduate, it has to shed off after delivery

ACTIVELY

(Active management of 3rd Labor)

AMTSL

if AMTSL = 5-10 min

↓

Bleeding ↓

↓

Chances of PPH ↓

↓

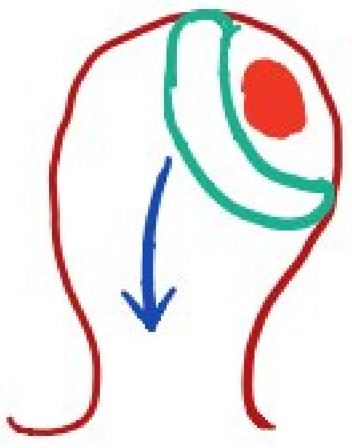
Maternal mortality ↓

Preffered Management = AMTSL (Best method to prevent PPH)

- Main event in 3rd stage of Labour is ⟶ Delivery of Placenta

Can occur by 2 methods

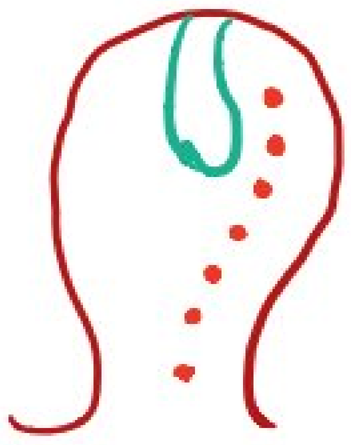
SCHULTZE METHOD



- Placenta starts seperating from center
- Retroplacental clot is formed. (Hemostatic)

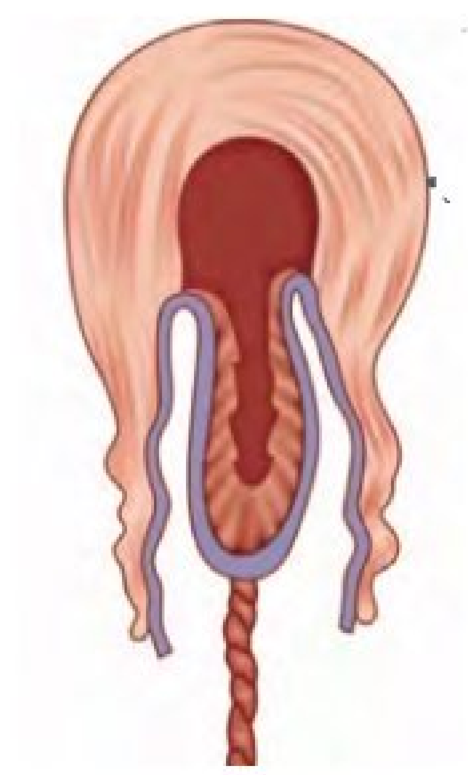
⇓

DUNCAN METHOD



- Placenta starts seperating Periphery.
- No retroplacental clot is formed

⇓

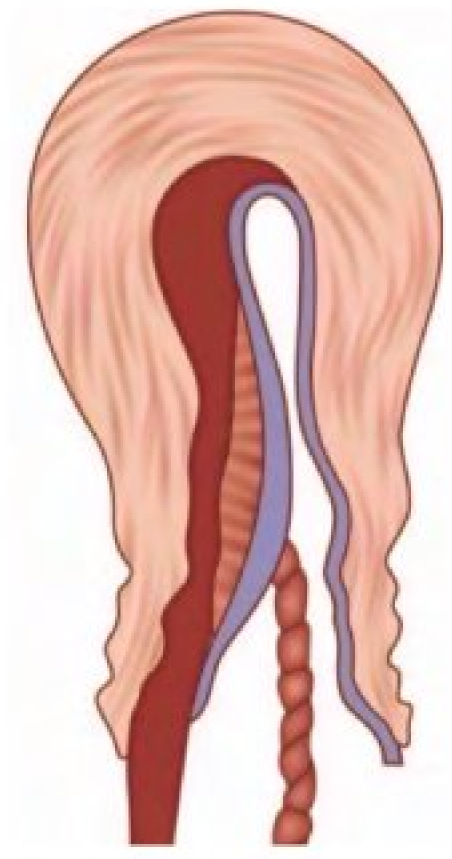


Blood Loss is less

- Bleeding is evident only after entire placenta has seperated
- Shiny fetal side of Placenta comes first

S → Shiny
S → Schuntze

- MC Method



Blood loss is more

- Bleeding is evident as soon as placenta seperates
- Dull maternal side of Placenta which comes first

D → Dull
D → Duncans

- Less common

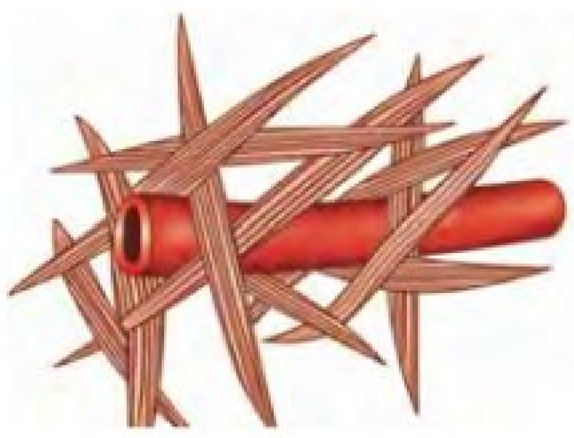
IMPORTANT ONE LINERS

- Most important factor responsible for Placental seperation is Uterine contractions.
- In all cases of Retained placenta, 1st management is ↑ uterine contraction by giving oxytocin (But Methergin, PGs are not recommended)

If even after giving oxytocin, if placenta doesn't getting seperated
↓
Manual Removal of Placenta (done in OT under GA)
↓
if can't find plane b/w Placenta & Uterus
↓
MORBIDLY ADHERANT PLACENTA (Rx: Hysterectomy)

- Line of seperation of placenta lies along → Zona Spongiosa

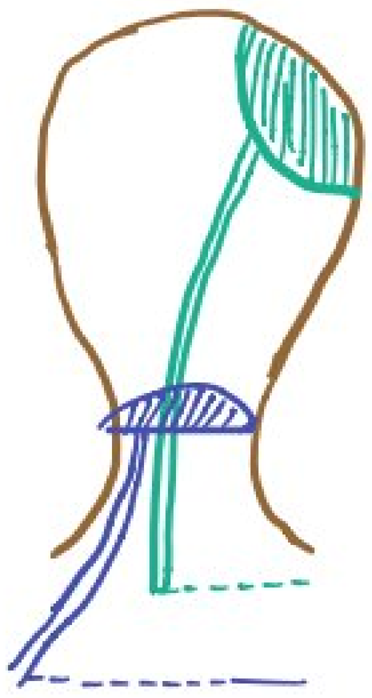
Living Liquature : Middle layer of myometrium, it has fibers in criss-cross manner in which blood vessels are there in b/w them
↓
When uterus contracts
↓
Blood vesels constricted



∴ Atonic uterus is MCC of PPH

- Height of uterus immediately after delivery → Just below the umblicus (~20 wk of pregnancy)

SIGNS OF PLACENTAL SEPARATION



i. Sudden gush of blood per vagina
ii. Supra pubic bulge
iii. Height of uterus increases slightly
iv. Apparent Lengthening of cord

M.Important : Feel the placenta in vagina > Lengthening of cord.

STEPS IN AMTSL

1. Administer uterotonic agent within 1 min of delivery of baby (M.Important Step)
2. Delayed cord clamping
3. Deliver placenta by controlled cord traction / Modified Brandt Andrews technique
4. Intermittent assessment of uterine tone → done by doctor (Earlier → Uterine Massage)

STEP I → Injection of Uterotonic Agent

<table>
<tr><th>DRUG</th><th>DOSE</th><th>IMPORTANT POINTS</th></tr>
<tr>
<td>OXYTOCIN
↓
Only Drug Recommended by WHO = As the Ist Line drug to prevent PPH</td>
<td>10 IU, IM or IV infusion</td>
<td>·Natural oxytocin : Nona peptide
·Synthetic oxytocin : Octapeptide
·Natural oxytocin is synthesized by Paraventricular Nucleus of Hypothalamus
·It's stored in Posterior pituitary
·t½ ⇒ 3-5 mins
·Storage temperature ⇒ 2-6°C
·can be given ⇒
<table>
<tr><th></th><th>IM</th><th>IV infu.</th></tr>
<tr><td>onset of action</td><td>3min</td><td>Immediate</td></tr>
<tr><td>Duration</td><td>3hrs</td><td>1hrs</td></tr>
</table>
· Never given as IV Bolus
Leads to Hypotension & Cardiac</td>
</tr>
</table>

		Arrest ·Mechanism of Action 1. It causes influx of Ca^{2+} ions 2. It releases PGF-2α ⇓ Brings about physiological contractions ⇓ ·Upper uterine segments contract ·Lower uterine segments passively dilate · On & off contraction → this helps in maintaining blood flow to fetus ⇓ Can be used during labour to augment contraction · Leads to milk ejection reflex
METHYL ERGOMETRINE (Ergometrine, Methergin)	0.2 mg IM 	*M.O.A: Tetanic contractions ↓ can not be used at the time of Labour * It's phototoxic (vials are brown in colour) * In HIV → if used with protease inhibitor, it may lead to HTN * Never give methylergometrine IV Because it can lead to HTN CONTRAINDICATIONS T → After delivery of Ist twin O → Organic Heart disease

		P → Pre eclampsia } → HTN E → Eclampsia } R → Rh incompactibility
SYNTOMETRINE	5 IU Oxytocin + 0.5 mg Methylergometrine	* Not readily available * Expensive * Highly potent
CARBITOCIN (Donot confuse c̄ CARBOPROST)	100 mcg slow i/v	* Synthetic oxytocin * Octapeptide * t½ → 85-100 mins
PGE 1 : MISOPROST (Not best drug)	600mcg – oral	* S/E : Hyperthermia, chills ↓ Dose dependent * Can be used in asthmatics * C/I in previous cesarean section
PGF - 2α CARBOPROST (Best) Trade Name : HEMBATE Best drug to treat and prevent PPH	250 mcg IM	* S/E : Diarrhea * C/I in asthmatics & in suspected amniotic fluid embolism.

OFF LABEL ⟶ Not recommended by WHO

↓

DINOPROST : PGE2

↓

Dose : 20mg supository per rectum

STEP-2 ⇒ Delayed Cord Clamping

≥ 1min - 3min

⇓

* 80ml of blood in cord

(≅ 50 mg of Fe)

⇓

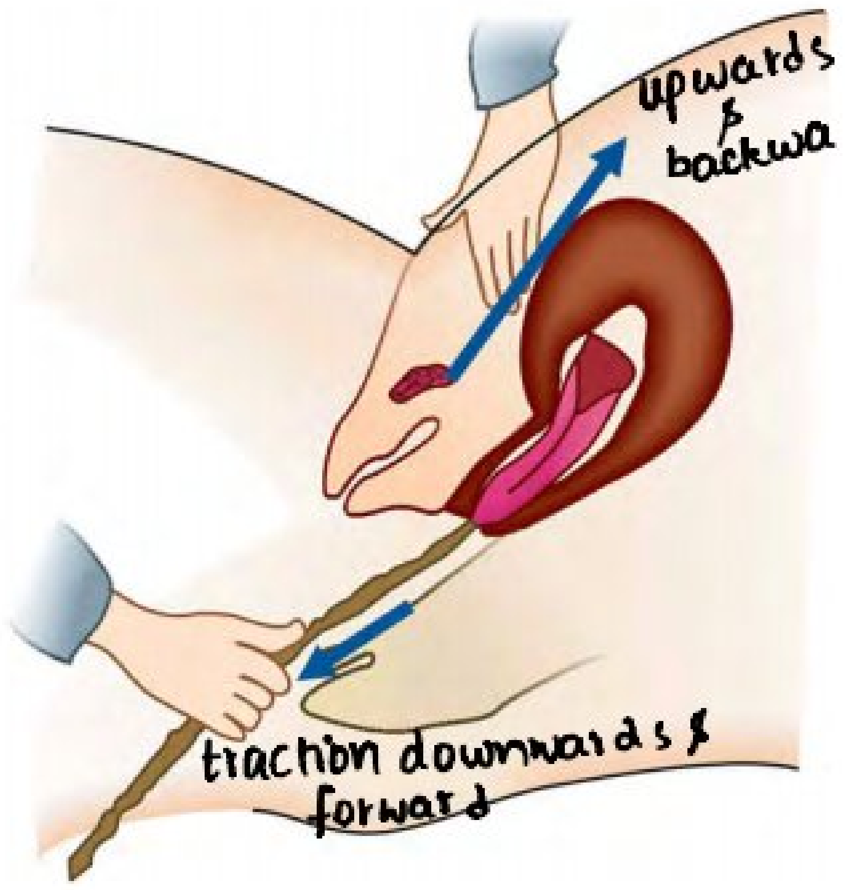
goes to baby (This is sufficient to prevent neonatal anemia)

EARLY CORD CLAMPING (< 1min)

1. Birth asphyxia
2. Neonate needs Recusitation
3. Baby is diagnosed case of Heart disease.
4. Rh negative Pregnancy
5. HIV +ve Female

→ Early cord clamping is not a part of ATMSL

STEP-3 – Delivery of Placenta by controlled cord traction :- (or) Modified Brandt Andrews technique



⇓

Make sure placenta is seperated

⇓

Other wise it can results in

⇓

UTERINE INVERSION

UTERINE INVERSION

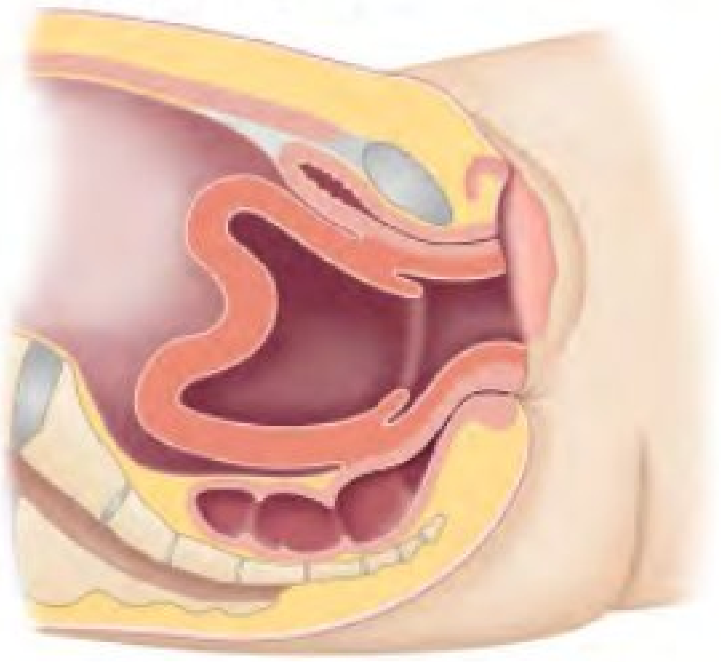
↓ Most Common Cause
↓
Mismanagement of 3rd stage of Labour

↓ Patient goes into shock immediatly
⇓
d/t Neurogenic Shock
↓
Later on there is Hemorrhagic Shock

↓ MCC of death in Uterine inversion
↓
Hemorrhagic Shock

DEGREES OF UTERINE INVERSIONS

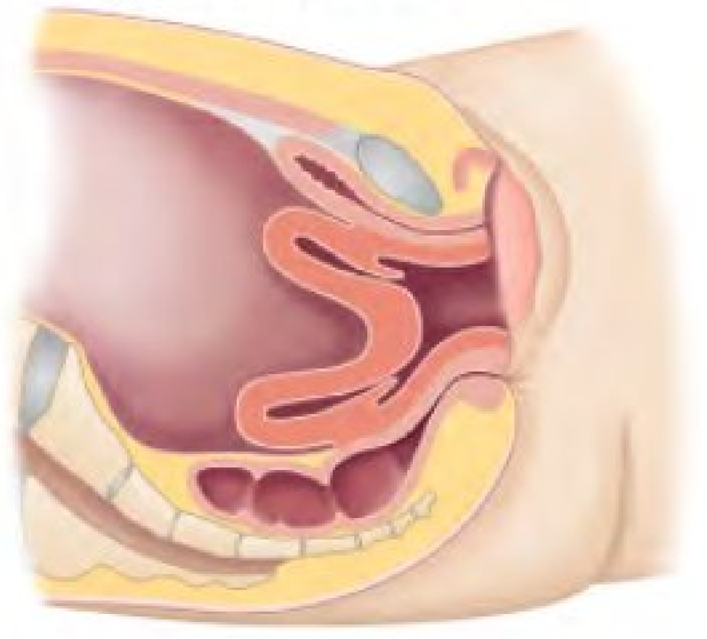
A 1st. degree inversion



1st Degree

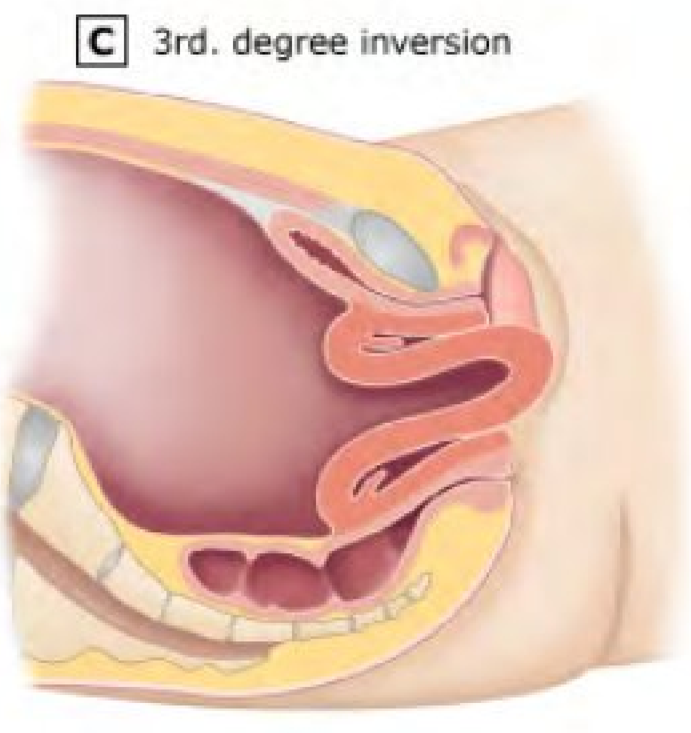
Fundus is with in Endometrial cavity above level of Internal os

B 2nd. degree inversion



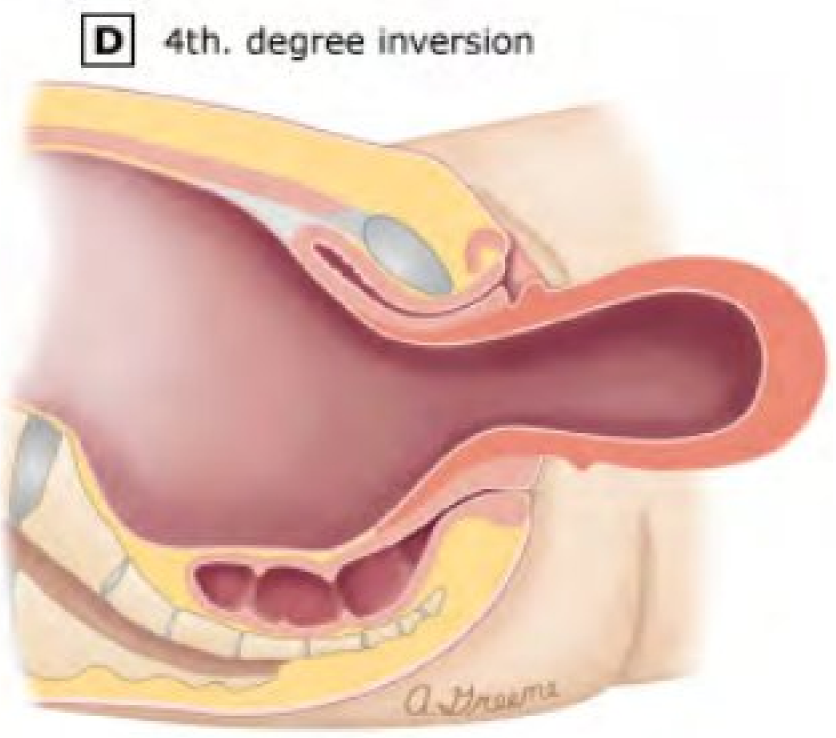
2nd Degree

Funds reachs uptil internal os (or) below os



3rd Degree

Fundus reaches uptil external os (or) below it.



4th Degree

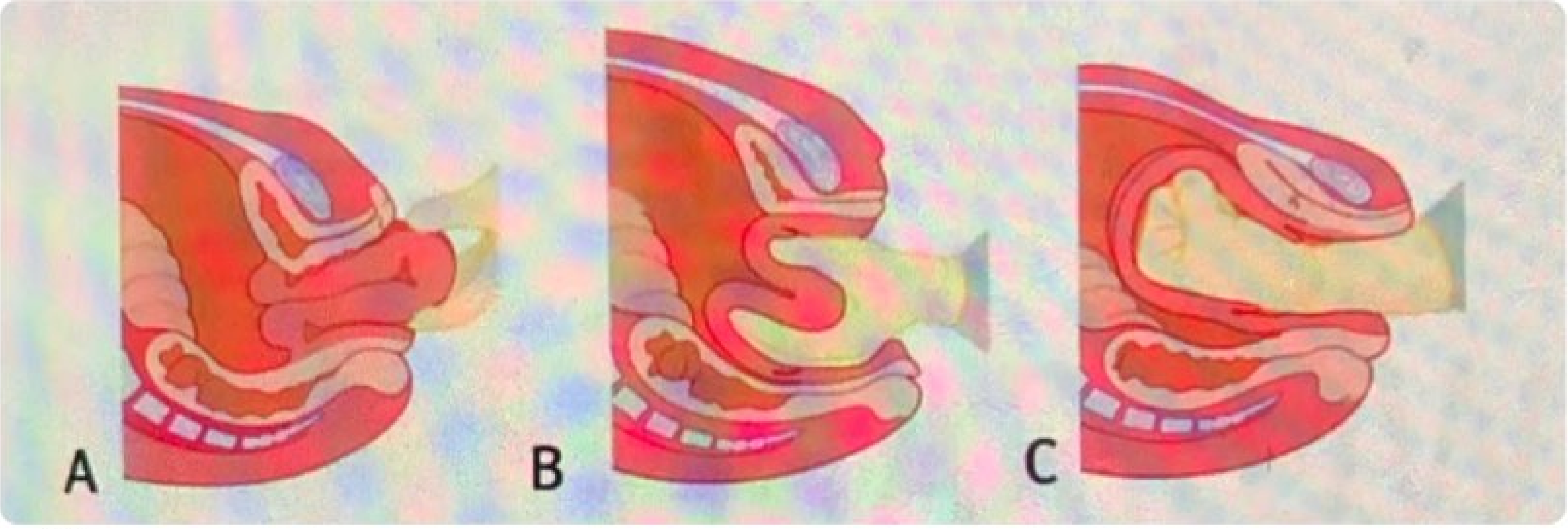
Entire uterus lying outside introitus

Another classification of Uterine Inversions

- i. Acute inversion → occurs within 24 hrs of delivery.
- ii. Subacute inversion → occurs after 24 hrs of delivery but c̄ in 1 month of delivery
- iii. Chronic inversion → ≥1 month of delivery

Management of Uterine inversion

1. Resuscitate the patient
2. Stop uterotonic drugs
3. Do not attempt Remove Placenta.
4. Correct inversion manually (JHONSON METHOD)



⇓

Unsucessful

Vitals of Patient Stable
↓
Immediate surgical management
↓

MOST COMMON : HUNTINGTON METHOD

Other methods :
- Haultain
- Spinelli
- Kustner

Vitals stable
⇓
Give uterine Relaxant (Terabutaline/ Nitroglycerine)
⇓
Try to Reposition uterus manually
⇓
Unsuccessful
⇓

Hydrostatic Method

(O Sullivan Hydrostatic Method)

⇓

Unsuccessful

↑ do surgery

* Whenever correction of inversion is succesful ⟶ Start uterotonic drug ⟶ Await spontaneous expulsion of placenta

3 Important Questions :

1. Patient after delivery goes into stroke → Most probable cause : PPH
2. Patient goes into shock immediately after delivery → Most probable cause : Uterine Inversion
3. Patient goes into unexplained shock after delivery → Most probable cause : Amniotic Fluid Embolism

STEP 4 : Intermittent access of uterine tone

Not a part of AMTSL :

1. Early cord clamping
2. IV Ergotmetrin
3. Uterine massage +/-

PPH

- Normal blood loss in vaginal delivery → <500ml
- Normal blood loss in cesarean section → <1 Litre
- Normal blood loss in twin delivery → < 1 Litre
- Normal blood loss in cesarean hysterectomy → <1.5 Litre

- In any case blood loss more than expected values in 24hrs of delivery or upto 12 weeks of delivery
- ACOG : Blood loss ≥1000ml irresptive of mode of delivery
- Bleeding is accomponied by signs & symptoms of hypovolemia, within 24 hrs of delivery irrespective of route of delivery

ATLS (Advanced Trauma Life Support) → Classification of PPH

- Class I : Blood Loss → <15%
- Class II : Blood Loss → 15-30%

- Class III : Blood Loss → 30-40%
- Class IV : Blood Loss → ≥40%

CLASSIFICATION OF PPH

PRIMARY PPH
↓
Occurs c̄in 24 hrs of delivery
↓
MCC : Atonic PPH

SECONDARY PPH
↓
Occurs after 24 hrs but before 12 weeks of delivery
↓
MCC : Retained Placental tissue

*Overall MCC of PPH : Atonic PPH

CAUSES OF PPH - 4 Ts

↓ Tone of uterus - MC
Trauma - 2nd MCC
↓ Thrombin (Bleeding disorders)
Tissue (Retained Placental tissue)

FACTORS PREDISPOSING TO ATONIC UTERUS

1. Overdistension of Uterus →
   - *Twins
   - *Polyhydroamnios
   - *Macrosomia (Diabetes)
   - *Grand Multipara (>5 delivery)
2. Prolonged Labor
3. Precipitate Labor (Time interval b/w onset of Labor to delivery of fetus = 3hrs)
4. Prolonged oxytocin
5. Chorioamnionitis
6. Previous h/o PPH
7. Antepartum Hemorrhage
8. Adherent Placenta
9. Fibroid Uterus → surface area of uterus ↑)

HTN DOES NOT LEAD TO PPH

Placental Anomalies that can lead to PPH :

1. Placenta succenturia
2. Placenta bilobata
3. Placenta spuria

MANAGEMENT OF PPH

1st STEP : Resuscitation of Patient

1. Secure 2 Large bore I/V lines
2. IV Fluids (Crystalloids)
3. Catheterize Bladder
4. Blood grouping, Hematocrit, Coagulation profile
5. Arrange Blood.

↓

Shock Index : Helps to determine to transfuse blood

↓

Shock Index = HR/SBP

- Normally → 0·5 to 0·7
- In pregnancy → 0·6 to 0·8
- If shock index = ≥ 0·9 to 1·1 → Indication for Blood Transfusion

2nd STEP : Distinguish b/w ATONIC PPH & TRAUMATIC PPH

on perabdominal examination

Uterus can't be palpated → ATONIC PPH

Uterus can be palpated → TRAUMATIC PPH

MANAGEMENT OF PPH

1st STEP : Give uterotonic drug

Drug recommended by WHO → OXYTOCIN

+

Simultaneously do Uterine Massage

- Dose of oxytocin → 30-40 IU as IV infusion in NS (or) RL

(Not in 5% dextrose, as in PPH oxytocin has to be given for a longer time, therefore we can't use electrolyte deficient media, it can lead to volume overload & Hypernatremia)

- If oxytocin is not available → WHO Recommends ⇒ Other drugs are,

1. Methyl Ergometrine :-
   - 0·2 mg i/v
   - Repeated → every 2-4 hrs
   - Maximum → 5 doses
2. Syntometrine :- 5 IU + 0·5 mg
   - 5 IU ↓ Oxytocin
   - 0·5 mg ↓ Methyl Ergometrin
3. PGE1 (MISOPROST) :- 800mcg, Sublingually
   (WHO Recommends per rectal use of misoprostal, per rectal dose = 1000 mcg)
4. CARBOPROST (PGF-2α) :-
   - 250 mcg, i/m
   - Repeated → Every 15-90min
   - Maximum → 8 doses (i.e 2mg)^{Q}
5. Inj. Tranexamic acid : 1 gm i/v (slow over 20min)

Remember →

For treating PPH, Recomended by WHO;

1st Drug → Oxytocin c̄ simultaneous uterine massage & give inj. Tranexamic acid.

⇓

Bleeding doesnot stops

⇓

Methyl Ergometrine

⇓

BEST DRUG :→ CARBOPROST

Medical management should be tried for 30mins

⇓

if it fails

↓

DO MECHANICAL METHODS

MECHANICAL METHODS

- Recommended by WHO
  1. Uterine massage
- Not recommended by WHO
  - Uterine Packing

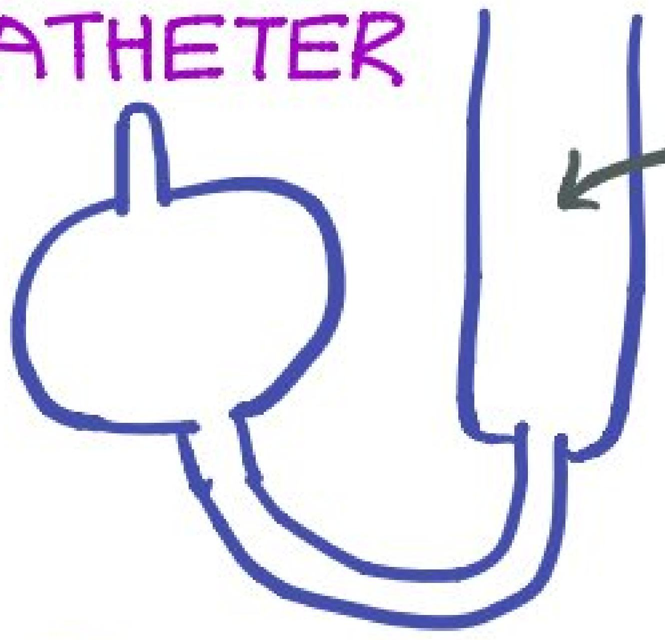
2. SENGSTAKEN- BLAKEMORE ESOPHAGEAL CATHETER

OR

BAKRI BALLOON CATHETER

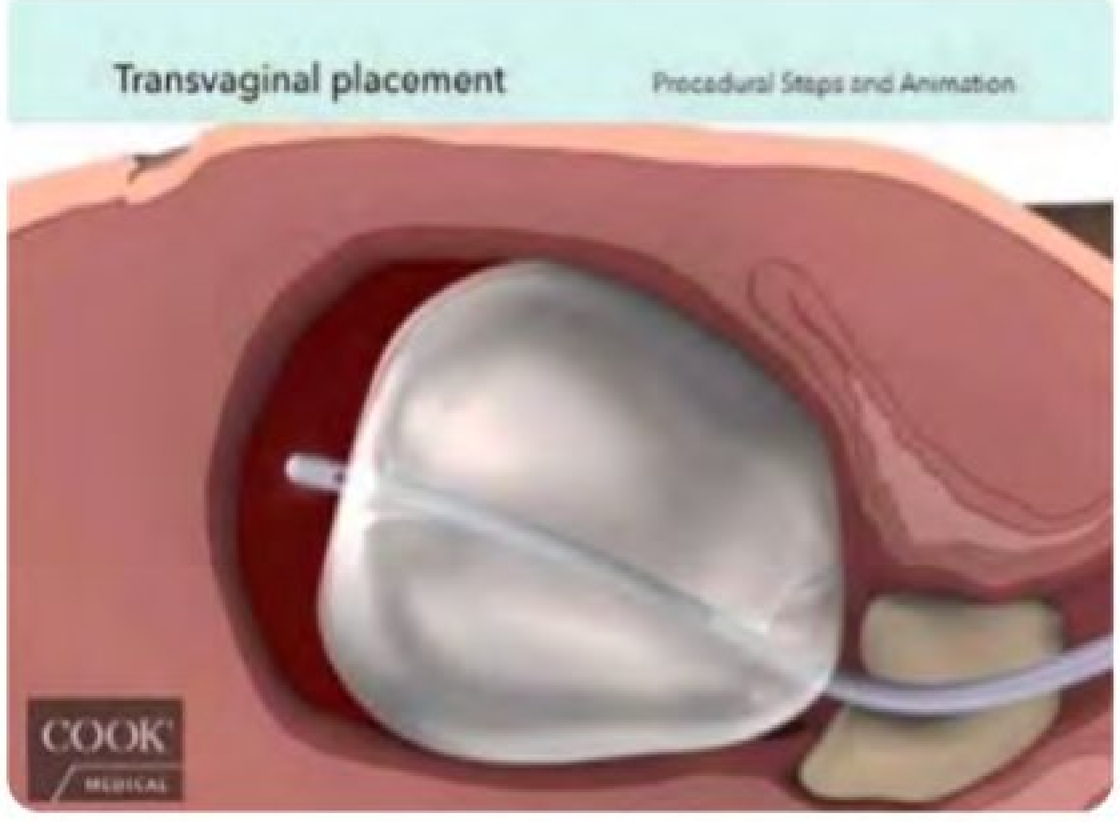
OR

CONDOM CATHETER



Fill it with NS → 300-500ml

Max capacity of Bakri Balloon : 500ml



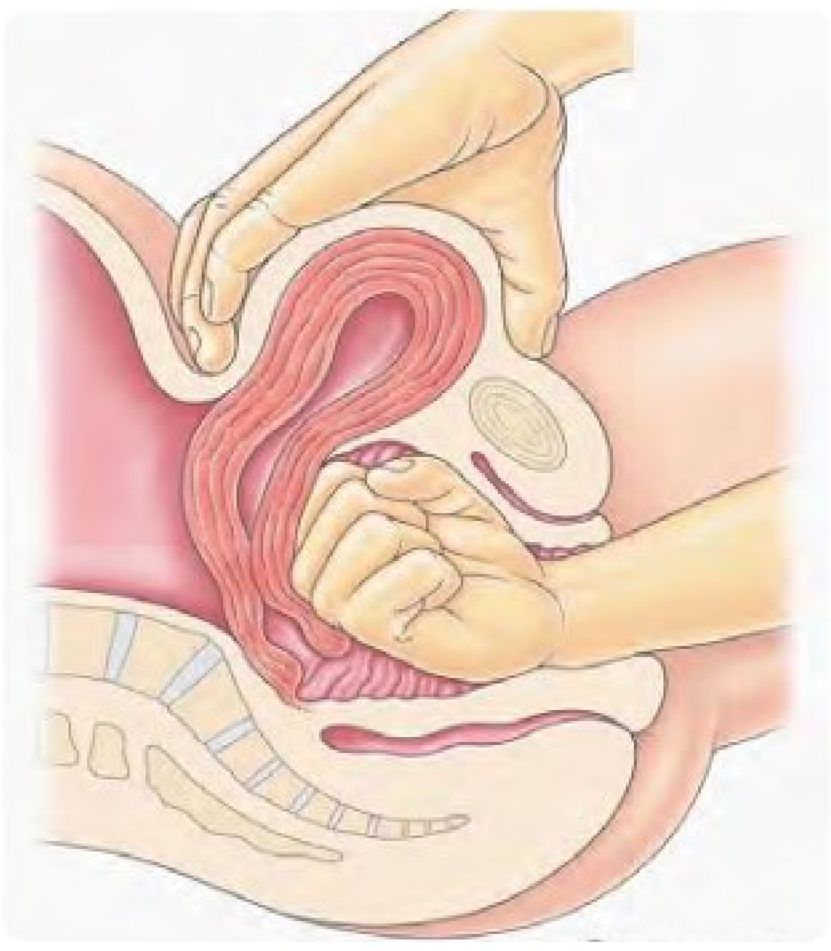
Bakriballoon

TEMPORARY METHOD RECOMMENDED BY WHO FOR T/T OF PPH

These are done as we wait for help or senior doctor to come (or) in transit to higher center.

1. Bimanual compression of uterus
2. External Aorta Compression
3. Use military anti-shock garments/treatment.

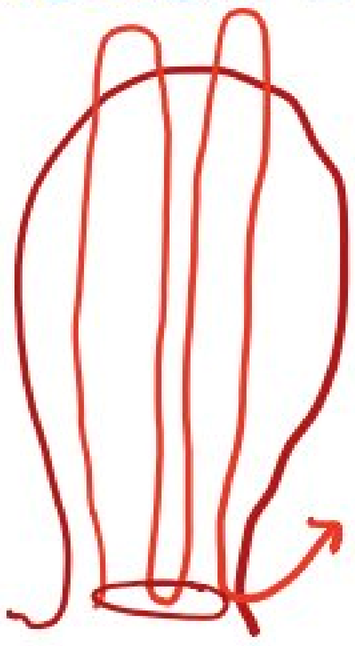
If mechanical methods fails → go for surgical method



Bimanual compression of ueterus

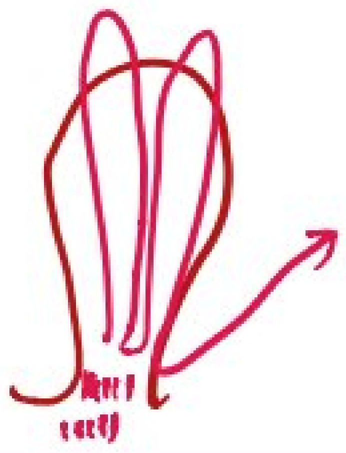
SURGICAL METHODS

1. Compression Sutures :- B Lynch sutures (MC)



OTHER COMPRESSION SUTURES ARE

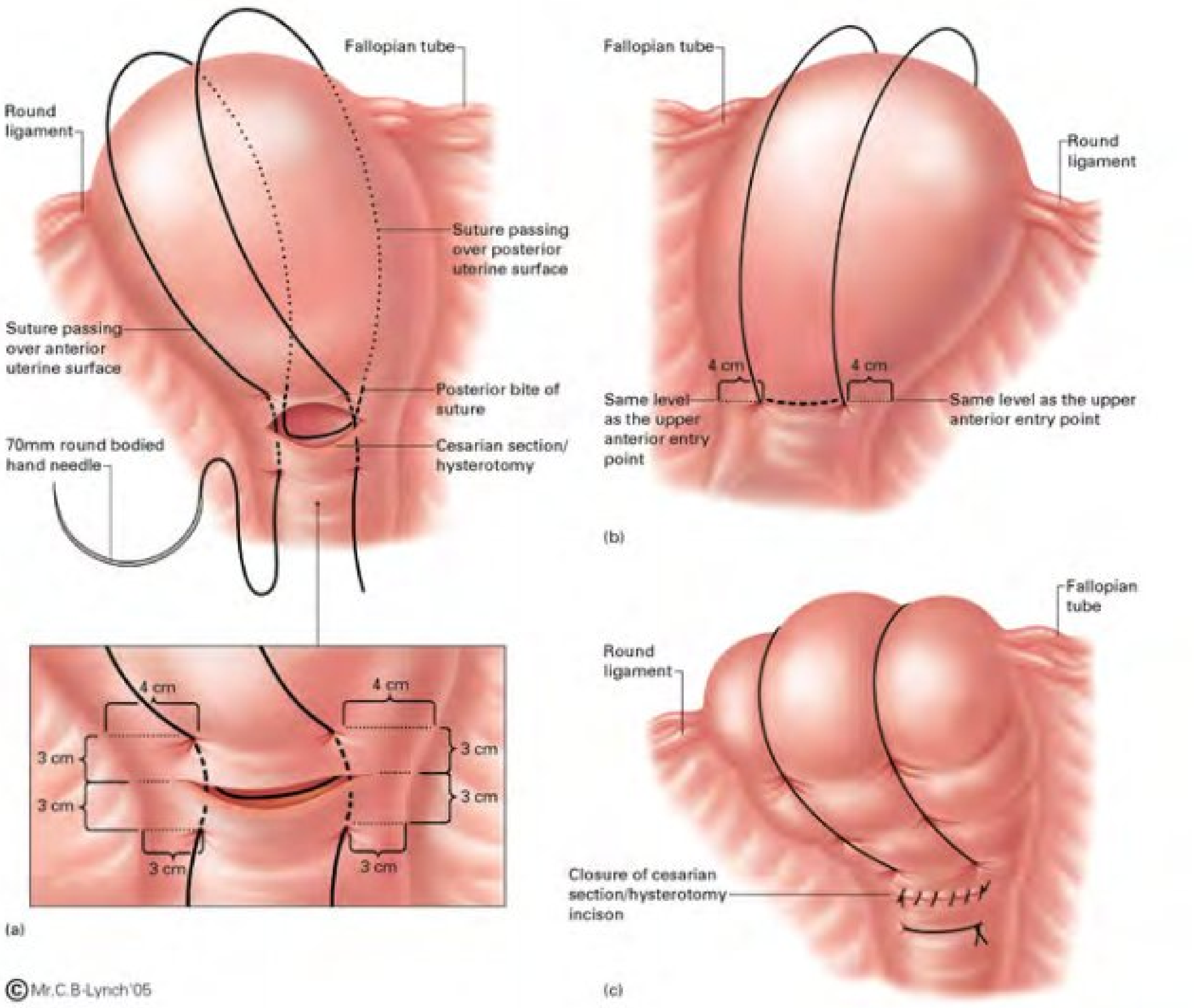
1. HAYMAN SUTURE



No need to open uterine cavity.

2. CHO SQUARE SUTURES

Sutures in form of square


Fallopian tube
Round ligament
Suture passing over posterior uterine surface
Suture passing over anterior uterine surface
Posterior bite of suture
70mm round bodied hand needle
Cesarian section/ hysterotomy
4 cm
4 cm
3 cm
3 cm
3 cm
3 cm
3 cm
3 cm
(a)
©Mr.C.B-Lynch'05
Fallopian tube
Round ligament
4 cm
4 cm
Same level as the upper anterior entry point
Same level as the upper anterior entry point
(b)
Fallopian tube
Round ligament
Closure of cesarian section/hysterotomy incison
(c)

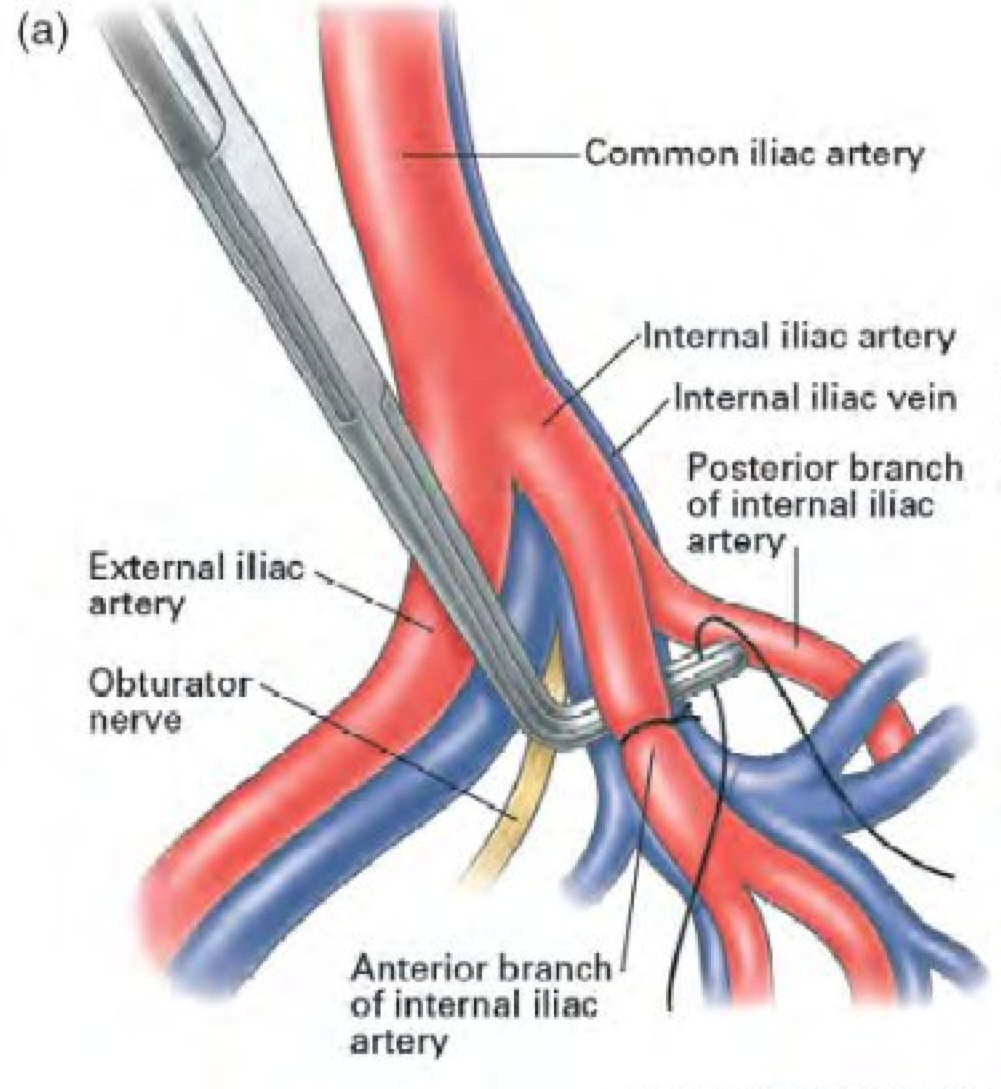
B lynch suture procedure

2. B/L Ligation of arteries which supplies uterus :
↓
* Uterine artery
* Ovarian artery
* Anterior division of internal illiac artery → *Site: 5cm below bifurcation of common illiac artery
* Pulse pressure ↓ by 80%

↓

Posterior division → Supplies lowerlimb

POSTPARTUM HEMORRHAGE



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↓

If bleeding still not getting controlled

⇓

Preffered { SUBTOTAL HYSTERECTOMY/SUPRACERVICAL HYSTERECTOMY
(Removal of only uterus, not cervix)

2nd BEST → TOTAL ABDOMINAL HYSTERECTOMY (TAH)
(Removal of Uterus + Cervix)
↓
If Bleeding still not getting controlled
↓
Apply Pelvic Pressure Packs
↓ ↓
UMBRELLA PACK PARACHUTE PACK

IMPORTANT QUESTIONS

- Delivery of fetal head during Normal vaginal delivery is Extension
- In breech and face presentation it occurs by Flexion
- MC position at the beginnig of Labor : LOT > LOA
- Occiput moves by 2/8 of a circle in LOT position & shoulders by 1/8th of a circle
- After external Rotation face of baby face towards right of thigh of mother & occiput towards the original position from where labor began.
- Time for giving Epistomy after crowning has occured.

Q According to WHO Guidelines, which of the following is true about Management of 2nd stage of Labour

a) Epistomy should be given as Routine
b) Warm cloth should be applied to perineum to prevent Trauma
[✓ c)] Manual support of perineum to maintain continous deflexation of head.
d) Delivery should be done in Lithotomy position.

Peurperium

PUERPERIUM :-

- period after delivery, upto 6wks postpartum

IMMEDIATE :- within 24 hrs of delivery

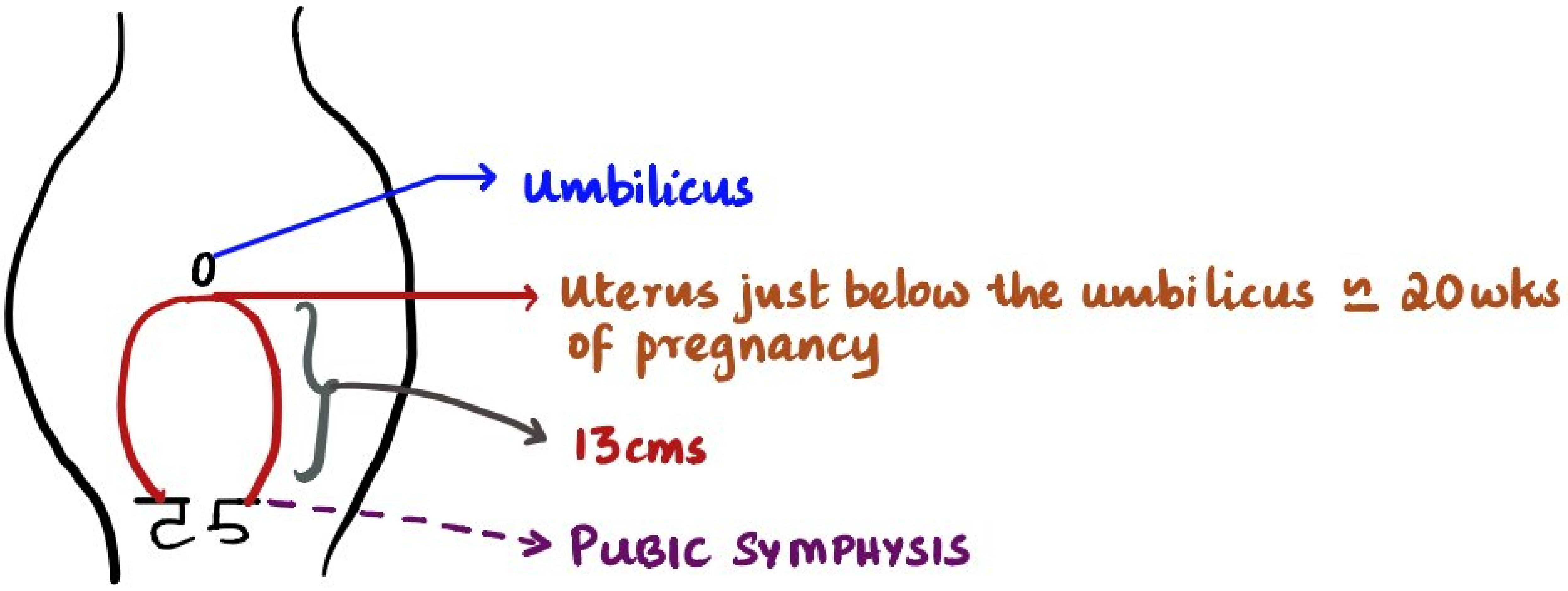
Early 24hrs to 1st wk of delivery

Late 2nd wk to 6th wk

- during this period, more chances of infections ⊕

INVOLUTION :-

1. UTERUS : position



- till 48 hrs uterus remains in this position

↓

- Starts decreases by 1.25 to 1.5 cm/day

↓

- lies at the level of pubic symphysis by 10-12 days after delivery.

SIZE OF UTERUS	WEIGHT OF UTERUS
Immediately after delivery	1000gm
1st wk	500gm
2nd wk	300gm
3rd wk	100gm
6th wk	60gm

CLINICAL SIGNIFICANCE : TUBECTOMY

within 1wk of delivery
↓
Post Partum Sterilization

After 6wks of delivery
↓
Interval sterilization

METHODS OF STERILIZATION :-

LAPAROSCOPIC STERILIZATION
- cannot be done in post partum period.

MINILAPAROTOMY
- can be done in post-partum period

(MC) method for female sterilization - LAPAROSCOPIC STERILIZATION

(MC) method of Post partum sterilization - MINILAPAROTOMY

SUBINVOLUTION : If uterus doesnot return to its normal position and size in required time frame

- **causes** - 1. Infection ⟶ ENDOMETRITIS
  2. Retained products of conception
  3. Fibroid uterus.
  4. Over distented uterus

- **Investigation** - USG + Gentle curettage + Antibiotics.

(MC) cause of ASHERMAN Syndrome - curettage done in post partum period.

ENDOMETRIUM: Endometrium of pregnancy ⟶ called as DECIDUA

Superficial layer :
sheds k/a LOCHIA

- Ⓝ seen → till 15 days after delivery
- can be seen → 24-36wks after delivery
- Fishy Odour
- Initially ALKALINE but towards end it is ACIDIC

Deep Layer
- This is responsible for Regeneration of Endometrium
- It begins by ⟶ 7days and completed by → 10 days
- Restored at 16 days
- Except : place where placenta was attached
  ↳ occurs by 6wks

LOCHIA RUBRA (RED)	RBC (Maternal)	1-4 days
LOCHIA SEROSA (YELLOW)	Mucus + WBC + Exudates	5-9 days
LOCHIA ALBA (WHITE)	Decidual cells	10-13 days

ENDOMETRITIS

- (MC) site : Placental site
- (MC) Manifestation : Puerperal pyrexia (100.4°F)
- (MC) cause of Puerperal Pyrexia : Endometritis
- (MC) organism : Group A Streptococci
- (MC) organism for Late onset of Endometritis : Chlamydia
- Apart from this → in most of the cases → MIXED Infection
    - ↳ Gram Negative : E.coli, Klebsiella
    - ↳ Anaerobic
- (MC) / Highest Risk of Endometritis : CESAREAN Section → 13 times more chances of Endometritis compared to vaginal delivery
- Other Risk Factors :
    1. Iron deficiency Anemia
    2. Prolonged labour
    3. PROM
    4. Bacterial vaginosis
- (MC) Route spread → DIRECT SPREAD
- SIGNS : 1. Fever 2. Uterine tenderness/Fullness
- TESTS : WBC ↑ , ESR ↑ , C-Reactive Protein ↑
- Management : Broad Spectrum Antibiotics [GENTAMICIN + CLINDAMYCIN
  Or
  AMPICILLIN]

CHANGES IN CERVIX AND VAGINA:

CERVIX

- External OS

Nulliparous	Multiparous
↓	↓
o	⬭
Pinpoint/ Circular	Transverse/ Slit

- Transformation Rebuilds
  ↓
  during this period female has maximum chance of getting infection with HPV

∴ Intercourse should be avoided till 6wks after delivery.

VAGINA

- comes back to its Normal size and shape
- RUGOSITIES ⟶ appears in 3wks
- BIRTH TRAUMA is important Risk Factor for ⟶ PROLAPSE.

BREAST

- Milk secretion ⟶ PROLACTIN
- Milk Ejection ⟶ OXYTOCIN

Contraindications for Breast feeding

Maternal
- Alcohol
- i.v drug users
- Breast cancer
- Anti-Cancer drugs
- Infections
  - Active Herpes lesion on Breast
  - Active / untreated TB

Baby
- Lactose intolerance
- Galactosemia

PROLACTIN

- maximum levels are seen during } PREGNANCY

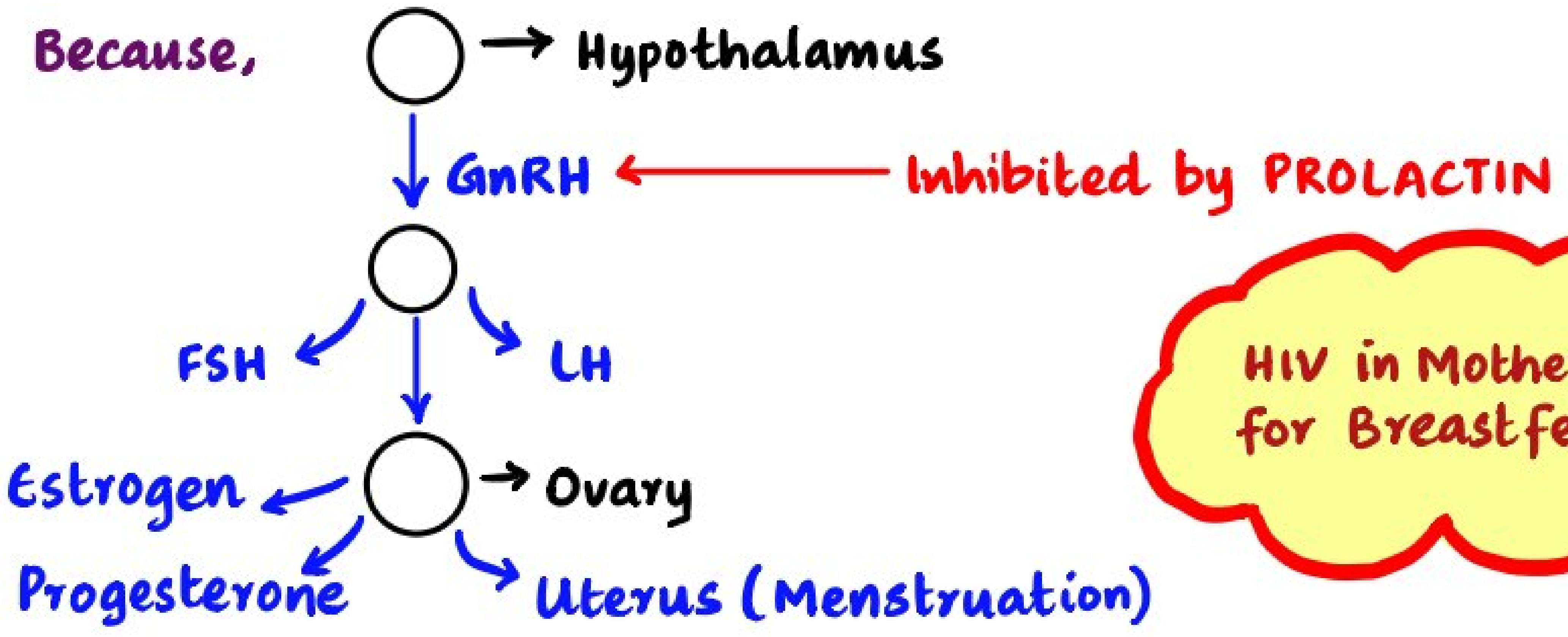
Why is there no Milk secretion during pregnancy???

- Because during pregnancy levels of Estrogen and Progesterone are HIGH

→ These have inhibitory effect on action of Prolactin
∴ There is NO MILK SECRETION during pregnancy but immediately after delivery when placenta is out level of Estrogen and Progesterone falls ⟶ inhibitory effect on Prolactin gone and milk secreted.

- ↑ Prolactin leads to AMENORRHOEA.

Because,



HIV in Mother is NOT a C/I for Breastfeeding

- DOC for ↑ Prolactin : CABERGOLINE
- DOC for ↑ Prolactin in INFANTILE FEMALE : BROMOCRIPTINE → Because it is also an OVULATION INDUCING DRUG.

Less Milk secretion ↓

GALACTOGOGUES :
- Metoclopromide
- Nipple stimulation
- Breast pump

suppresing milk secretion ↓

DOC : CABERGOLINE
- BROMOCRIPTINE
- Inj. MIXOGEN ↓ is not used now because of Risk of Venous Thrombosis

MASTITIS

* Infection of parenchyma of Breast ⟶ MASTITIS

* (MC) organism ⟶ STAPH. AUREUS

* (MC) Source ⟶ INFANTS NOSE + MOUTH

NOT A CONTRAINDICATION FOR BREAST FEEDING

* Symptoms ⟶ 3-4 wks

* It is unilateral

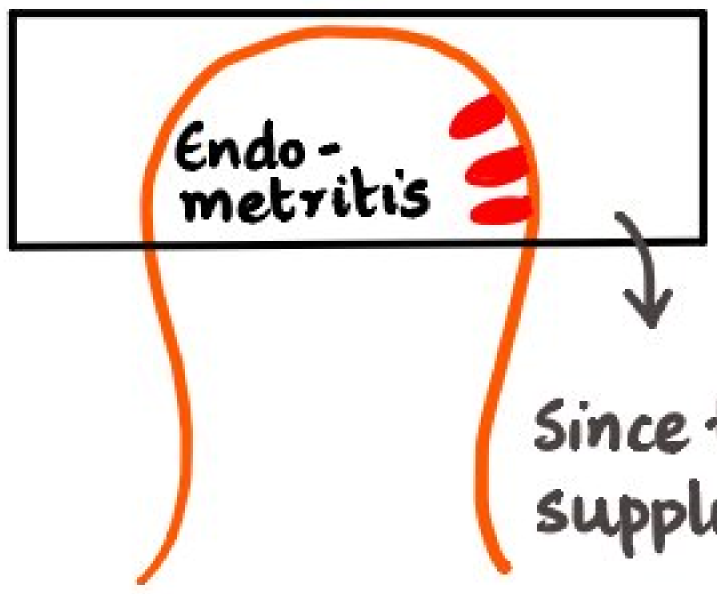
* Presentation : c/o FEVER, BREAST ENGORGEMENT, TENDERNESS ⊕

* Management : PENICILLIN / DICLOXACILLIN
+ Milk expression

* In 10% of cases → It can lead to "BREAST ABSCESS"
↓
O/E → Fluctuant mass ⊕ or
Fever not resolving inspite of Antibiotics

Management : Incision and Drainage
(↓ General Anesthesia)

PELVIC THROMBOPHLEBITIS



Since this part is supplied by → OVARIAN VEIN

(MC) vein ⟶ Ovarian vein

(MC) ⟶ (R) Side Ovarian vein

ENDOMETRITIS

* Lead to thrombus formation in venous circulation of mother

* Acts as a Nidus , attracts aerobic bacteria → THROMBOPHLEBITIS

Presentation : Spikes of High Grade Fever, inspite of Antibiotics +
pain Abdomen +
Tender mass near cornua of uterus

IOC : MRI and CT

Management : High dose of Antibiotics + Heparin

PSYCHOLOGICAL PROBLEMS (BPD)

POSTPARTUM BLUES	POST PARTUM PSYCHOSIS	POST PARTUM DEPRESSION
• s/i 50-60% Mildest	• <1%, Serious	• 10-20%
• Gradual Onset within 10 days of delivery.	• Sudden Onset, 10-14 days	• Gradual onset, 4-6 months
• Withdrawal Progesterone	• Antipsychotic drug	• changes in HPO adrenal axis
• Reassurance + Care		• SSRI - FLUVOXETINE

NOT BREASTFEEDING

Return of Menstruation ↓
40% → 6 wks
80% → 12wks

Return of Ovulation ↓
≃ 7wks
(5-11 wks)

CONTRACEPTION OF CHOICE IN LACTATING FEMALES

1st choice → IUCD → Copper IUCD

Time of Insertion

POST PLACENTAL
CuT 380A

POST PARTUM
↓
within 48hrs of delivery

If not done, then wait for 4-6wks

	NON BREAST FEEDING	BREAST FEEDING
LNG - IUCD	Immediately	After 4 wks
Progesterone Only Pills	Immediately	After 4-6wks

Combined Oral Pills	3wks	After 6months

BARRIER CONTRACEPTIVES : When she resumes coital activity (after 6wks)

QUESTIONS :-

Q. Post partum decidual secretions present are referred to as

a. LOCHIA b. Vasa previa

c. Bleeding P/v d. Decidua capsularis

Q. Lochia in correct order during puerperium :

a. Rubra, serosa, alba
b. Serosa, Rubra, alba
c. Alba, serosa, Rubra
d. Alba, mucosa, Serosa

Q. Likely size of uterus 8wks Post Partum is

a. 100gm b. 500gm c. 700gm d. 900gm

Q. Sarita, a 30yr old woman develops DVT in her left calf on 4th Post OP day following C-section done for fatal distress. The patient is started on Heparin and is scheduled to begin a 6wks course of Warfarin therapy. The patient is devoted mother who wants to breast feed her baby. What is the advise which is given to patient

a. Patient may continue breast feeding at her own risk.

b. Patient should breastfeed her baby only if her INR is at < 2·5

c. Patient can breastfeed her baby after 6wks course of warfarin is over

d. warfarin is not a C/I for Lactation

e. Warfarin is absolutely C/I during Lactation

Q. Decrease Lactation seen in all except

a. maternal anxiety b. Breast abscess

c. Metoclopropamide d. Bromocriptine

d. Cracked Nipple

Q. About colostrum true statements are all except

a. contains more Sodium
b. Rich in Immunoglobulins
c. contains more proteins
d. contains more fat

Q. Contraception of Choice in Lactating mothers :

a. Barrier method
b. Progesterone only pills
c. Lactational Amenorrhoea
d. OCP

Q. The uterus becomes pelvic organ after delivery in

a. 10 to 12 days
b. 12 to 14 days
c. 14 to 16 days
d. 16 to 18 days
e. 18 to 20 days

Post Term Pregnancy

POST TERM PREGNANCY : Any pregnancy which goes beyond 42wks (≥42wks)

NAEGLE'S FORMULA : 1st day of LMP + 9months + 7days → EDD

If 1st day of LMP = 1st Sept 2018 , EDD = 8th June 2019

* Only 4% of females deliver on exact EDD

* 50% of females deliver 1wk before or 1wk after EDD

Theoretically → Pregnancy can be carried maximum uptill 42wks
Beyond it, is called POST TERM PREGNANCY (>42wks)

POST TERM PREGNANCY

COMPLICATIONS

1. OLIGOHYDRAMINOS :
   - can lead to Meconium Aspiration Syndrome
2. MACROSOMIA
   - excessive fat deposited on shoulder and trunck region
     - a. Shoulder Dystocia
     - b. ↑ Cesarean Section
     - c. Intra Cranial Hemorrhage
3. PLACENTAL AGEING
   - placental insufficiency fetal distress

1st STEP

- Review her menstrual history

Fetal ACTH
↓
Fetal Adrenal Glands
↓
DHEA-S
↓
used by Placenta
↓
Synthesis Estrogen
↓
Signals to uterus to start contracting

∴ For labour to initiate → Intact Fetal Adrenal gland is required.

ANENCEPHALY can leads to POST DATED PREGNANCY.

- Management of Post Dated Pregnancy → INDUCE LABOUR

Q. Best tocolytic in Cardiac patient is

a. Atosiban b. Isoxsuprine

c. Nifedipine d. MgSO4

Q. On TVS which of the following shape of cervix indicates pre-term labour

a. T b. u c. Y d. O

T → Y → V → u

Q. Cut off value of cervical length at 24wks of gestation for prediction of Pre-Term delivery is

a. 0·5 cm b. 1·5 cm c. 2·5 cm d. 3·5 cm

Dose of Dexamethasone → 6mg 12hrly 4doses
Dose of Betamethasone → 12mg 12hrly 2 doses

Q. Anticipated Pre-Term Delivery Dose of DEXAMETHASONE given to mother is

a. 12 mg 12hrly 2 doses
b. 12 mg 24 hrly 2 doses
c. 6 mg 24 hrly 2 doses
d. 6 mg 12hrly 4 doses

Q. A woman comes with post dated pregnancy at 42wks. The initial evaluation would be

a. Induction of Labour b. Review of Pre-menstrual h/o

c. Cesarean section d. USG

Q. A Primipara complaints of sudden onset of vaginal discharge at 32wks pregnancy. P/A : NO CONTRACTIONS. Cephalic presentation. FHS → Ⓝ What is 1st Step in Management ?

a. Start Broad Spectrum Antibiotics
b. Start Corticosteroid
c. Perform Sterile Per speculum examination
d. Perform Sterile vaginal examination

Q. In Post Term Pregnancy, there is ↑ Risk of all except

a. PPH b. Meconium Aspiration Syndrome

c. Intracranial hemorrhage d. Placental insufficiency leading to fetal hypoxia

Q. Saffron coloured Amniotic fluid is seen in

a. Post maturity b. TB c. Breech d. Normal in Appearance.

ABORTIONS

- Definition : Pregnancy loss < 20wks. WHO : Pregnancy loss where conceptus wt. < 500g
- Ⓝ Fetus @ 20wks – 300g
- **SPONTANEOUS ABORTIONS** : Recurrent Abortion (Recurrent Preg. Loss) ≥ 3 consecutive losses

  or ≥ 2 Confirmed Pregnancy losses
- **RISK FACTOR** : ① ↑ Maternal age

  ② Previous h/o abortion : prev. 1 abortion = 20%
  prev. 2 abortion = 30%
  prev. 3 abortion = 40%
- (MC) cause of Spontaneous abortions ⟶ Chromosomal anomalies
  - 1st TM – 50%
  - 2nd TM – 30%

  ↓

  (MC) ANEUPLOIDY – AbⓃ in No. of Chromosome.

  ↳ TRISOMY (All trisomy grp) > Monosomy
- SINGLE (MC) cause of Spontaneous Abortion :

  MONOSOMY 'X'
  20% of all abortions

  $\xrightarrow{f/b}$ TRISOMY 16 : 16% of all abortions
  - most lethal Trisomy
  - most viable Trisomy is Tri. 21
- (MC) cause Recurrent abortion : Idiopathic > 50%
- (MC) cause in 1st / 2nd TM : APLA f/b Uterine AbⓃ
- Recurrent Pregnancy Loss : Evaluate her for 4 Established causes
  - ① APLA
  - ② Uterine AbⓃ
  - ③ Hypothyroidism
  - ④ Chromosomal Anomalies

I. CHROMOSOMAL ABNORMALITY – 4% cases

- Karyotyping is done for evaluation of RPL

II. HYPOTHYROIDISM – TSH evaluation done for all pregnant women.

↳ SUBCLINICAL can cause ABORTIONS

III. UTERINE ABNORMALITIES : can cause both 1st TM and 2nd TM Abortions

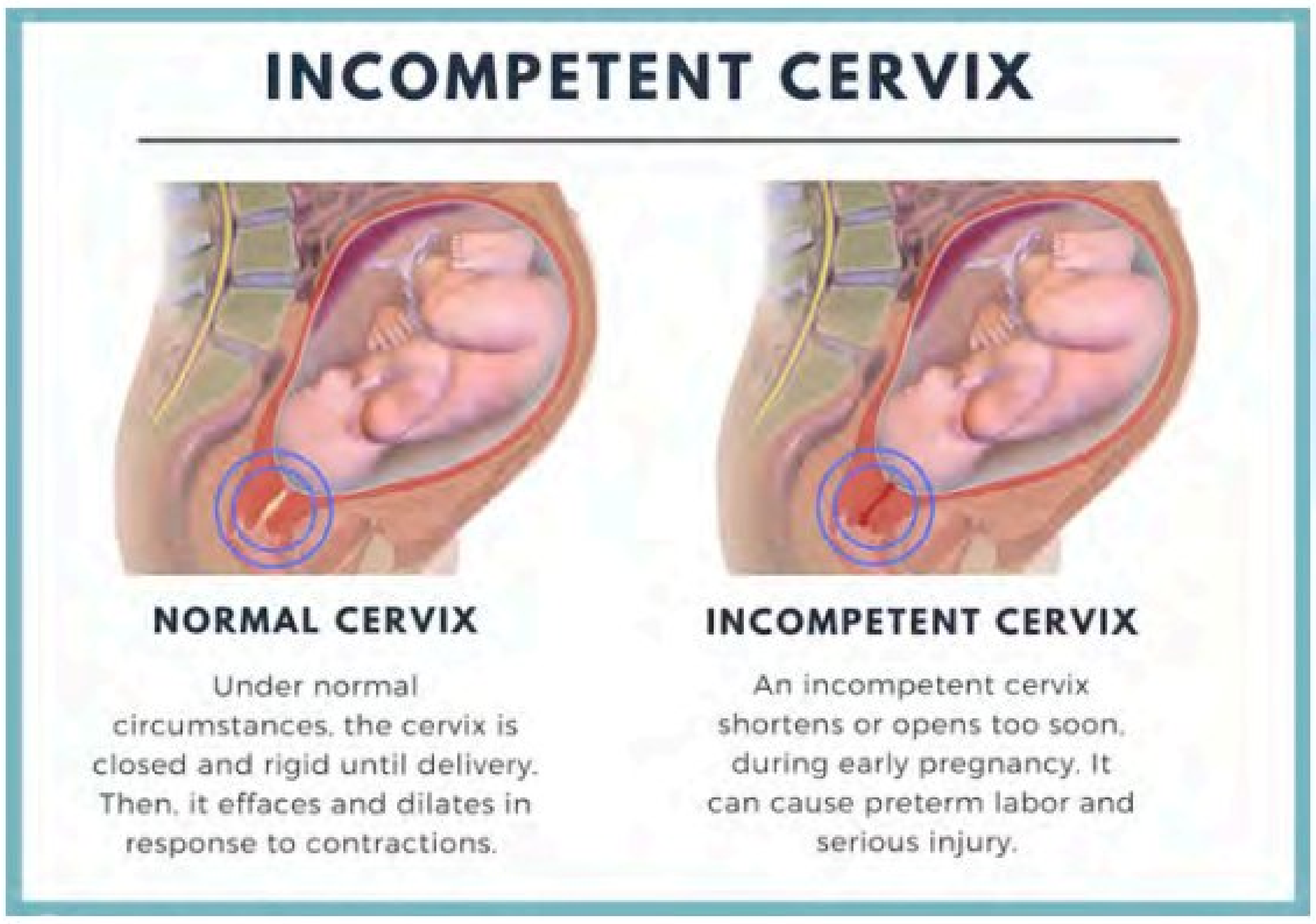
1. ACQUIRED – more common than CONGENITAL ANOMALIES

i) Cervical incompetence – (MC)

ii) FIBROID

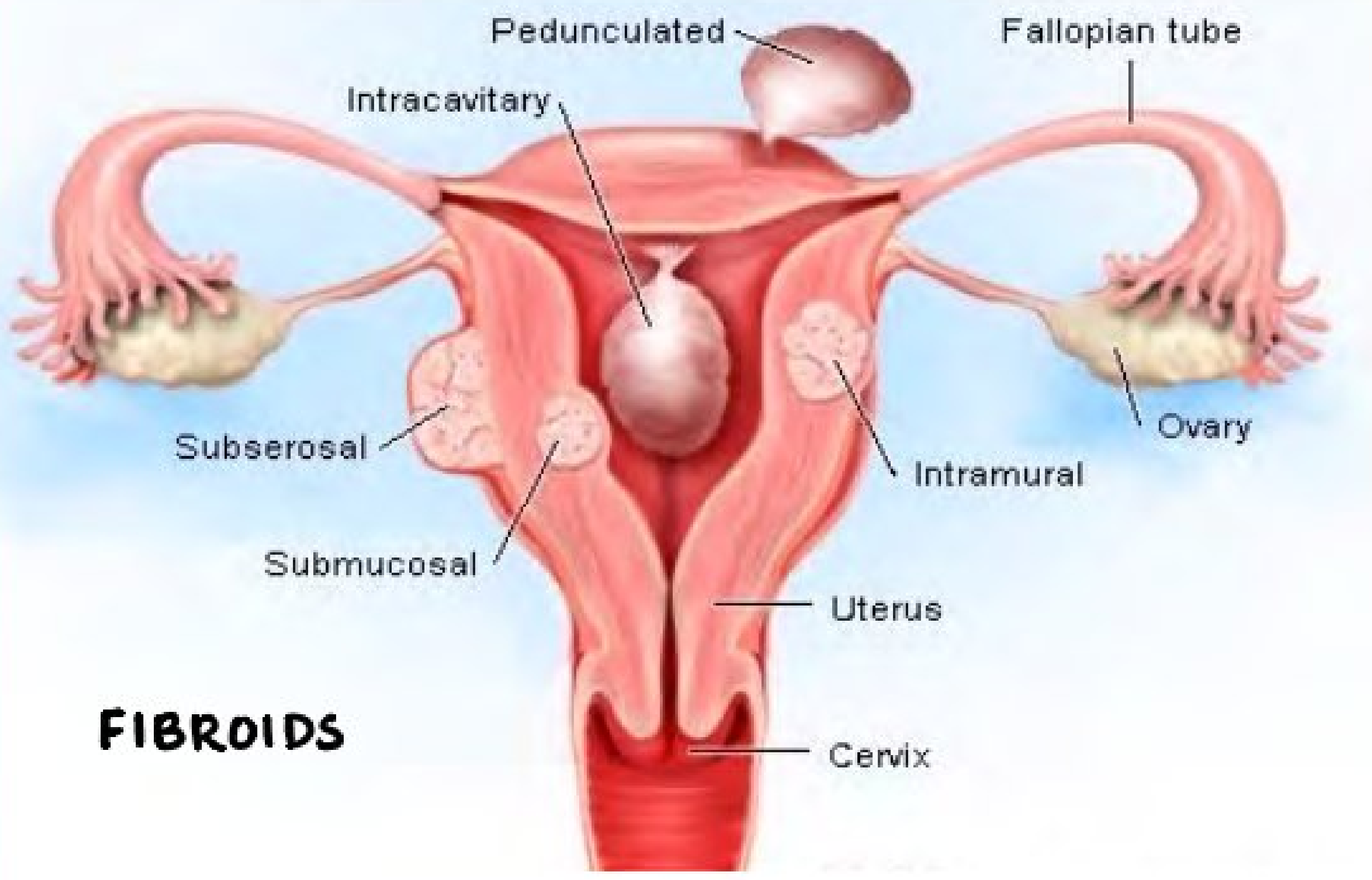
iii) POLYP

iv) ENDOMETRIOSIS



Under normal circumstances, the cervix is closed and rigid until delivery. Then, it effaces and dilates in response to contractions.

An incompetent cervix shortens or opens too soon, during early pregnancy. It can cause preterm labor and serious injury.



FIBROIDS

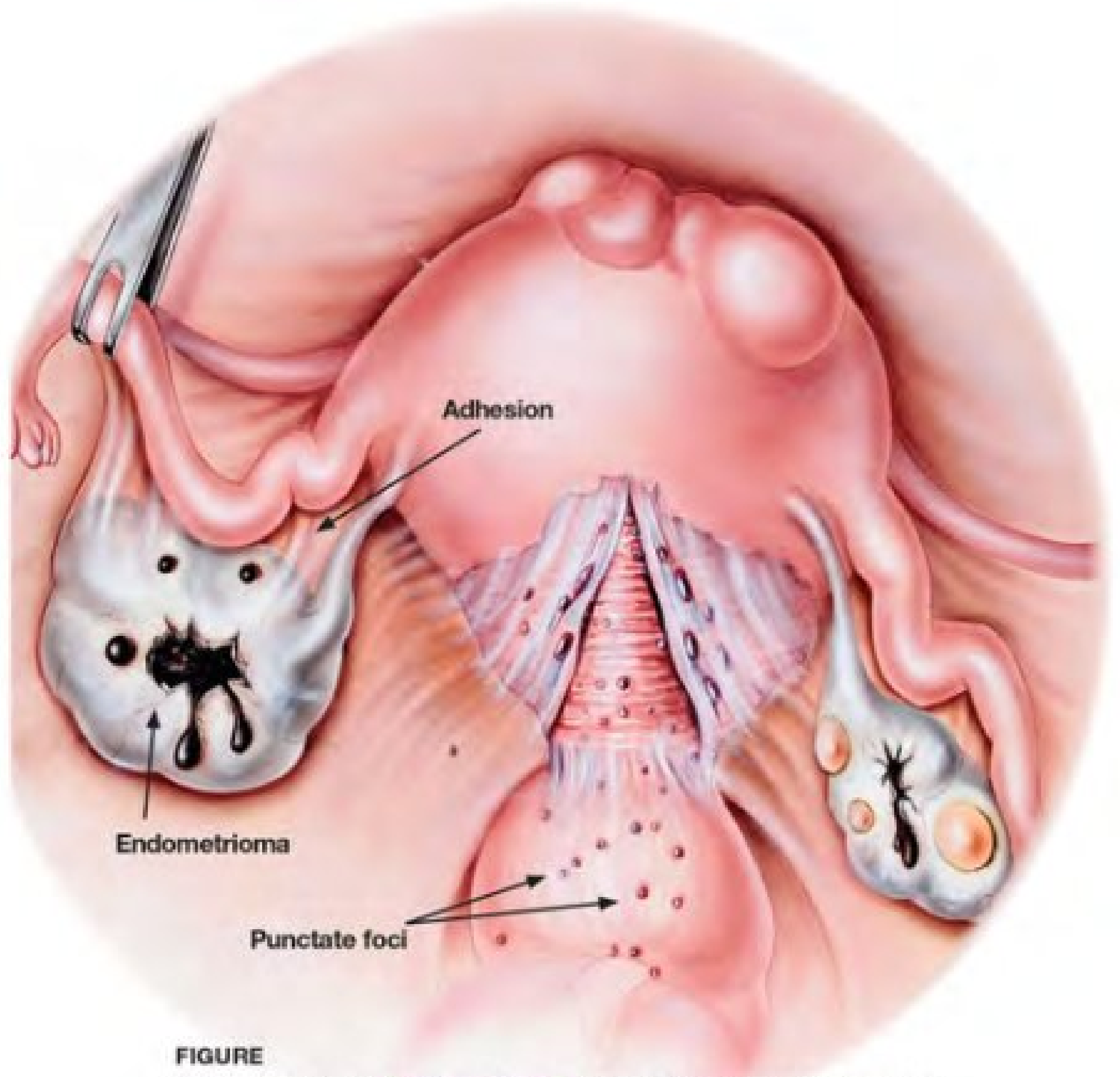
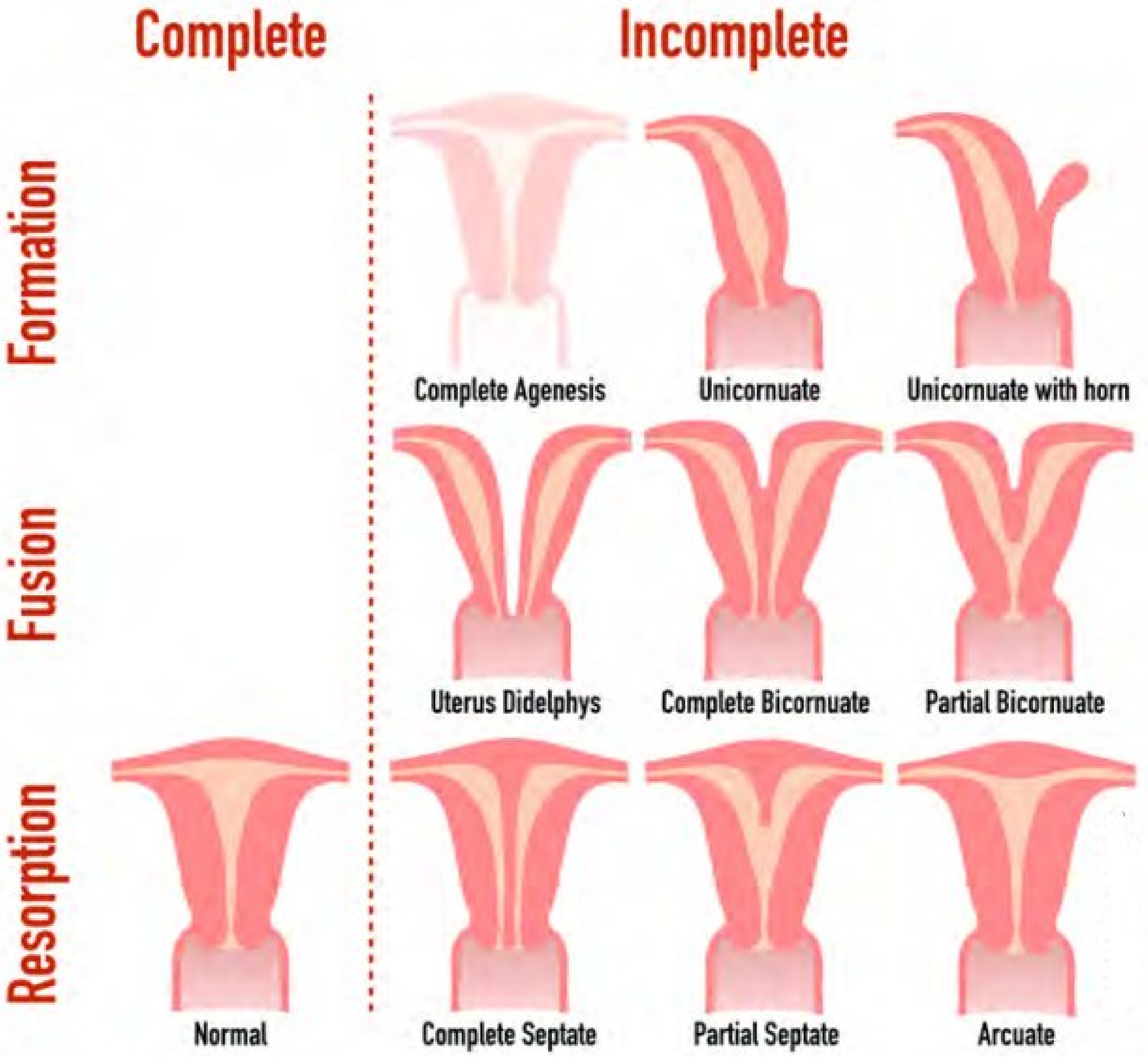


FIGURE
Of unknown cause, endometriosis—the displacement of endometrial tissue outside the uterus—results in lesions, usually found on the ovaries, fallopian tubes, and ligaments that support the uterus. When ovaries are involved, cysts called endometriomas may form. The surrounding tissue can become irritated, eventually developing scar tissue, adhesions, pain, and infertility.



2. CONGENITAL ANOMALIES

- (MC) Septate uterus
- Uterine AbⓃ cause 2nd TM ABORTIONS (characteristic)
- h/o RPL ? → To know about Uterine AbⓃ → Do USG [2D-USG] → NOT Diagnostic for Mullerian Anomalies
- IOC for MULLERIAN ANOMALY - 3D/4D USG ; Gold Std : MRI

CERVICAL INCOMPETENCE : weak Cervix unable to hold pressure of Pregnancy

- Diagnosis Based on 1. HISTORY , 2. USG

1. HISTORY BASED (characteristic h/o)
- Painless dilation of Cervix ; ≤ every pregnancy the POG of loss keeps increasing
- ≥ 2 consecutive
- Only in 2nd TM

2. USG BASED:

i) CERVICAL LENGTH: Most important ; Ideal Time - 2nd TM ; if it is < 25mm → Dx of Cervical Incompetence

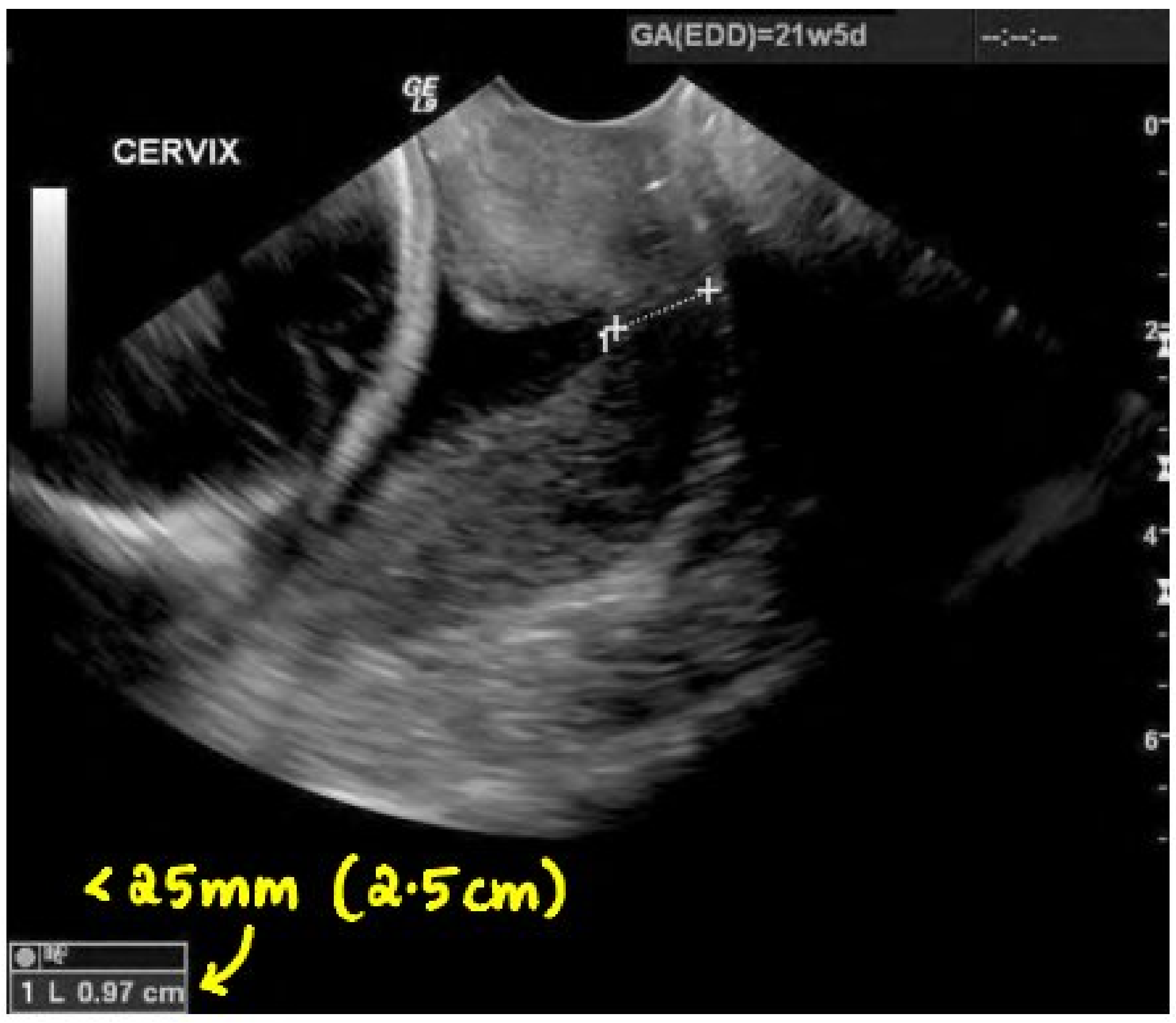
ii) INTERNAL OS Diameter: > 2cm in 2nd TM

iii) Relation of INTERNAL OS - CERVICAL CANAL Changes

Sequence

Ⓣ → Y → V → Ⓤ

Competent Cervix ← Ⓣ ... Ⓤ → Incompetent Cervix



< 25mm (2.5cm)

MANAGEMENT OF CERVICAL INCOMPETENCE

- CERVICAL CERCLAGE aka Cervical Stich + Progesterone

Ⓜⓒ used is McDONALD STICH aka PURSE STRING SUTURE

- Ideal time to put cerclage is 12-14 wks ; it can be put upto 24 wks.

Q. 3rd Time preg. Prev 2 painless abortion in 2nd TM i.e 14wk ⟶ Do CERVICAL CERCLAGE

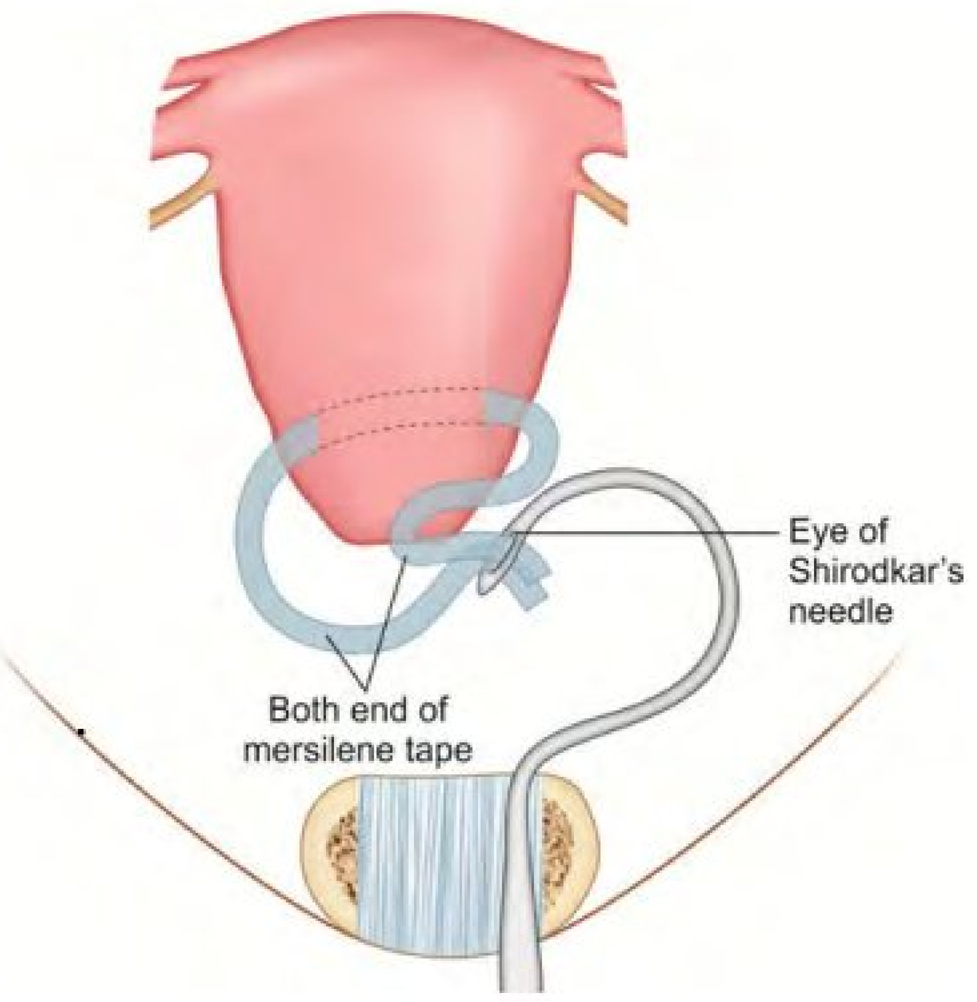
C/I for CERCLAGE

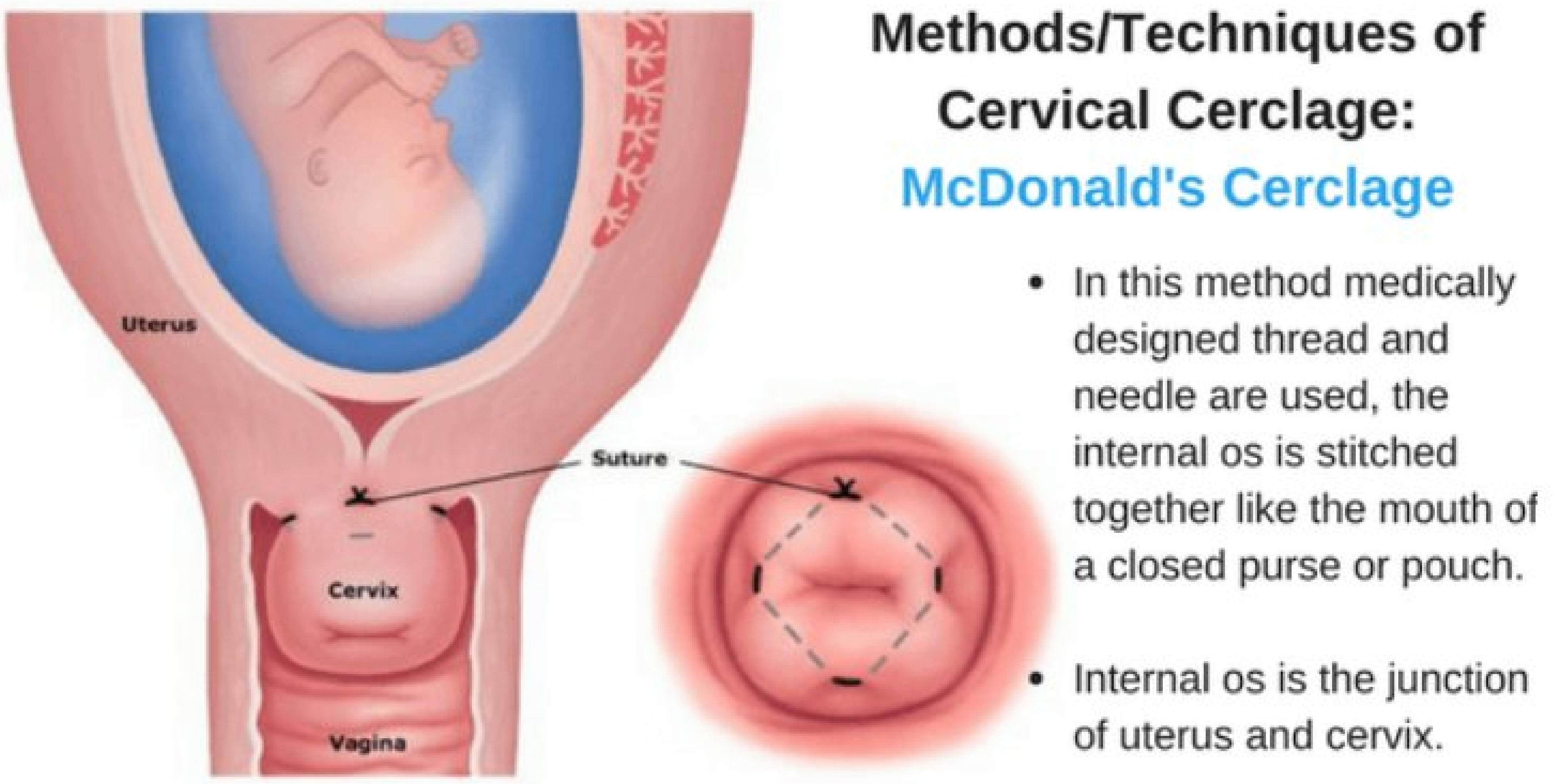
1. Memb. are RUPTURED
2. CURRENT PELVIC INFECTION
3. Fetus has GROSS CONGENITAL ANOMALIES

When do you remove Stich?

- done at 37 wks or anytime if these happens
  i) Rupture memb. eg. at 34 wks
  ii) Pt goes into Labour
  iii) Chorioamnionitis

SHIRODKAR'S TAPE - its MERSELENE Tape ; used if McDonald's Stitch Fails




Uterus
Suture
Cervix
Vagina

Methods/Techniques of Cervical Cerclage:

McDonald's Cerclage

- In this method medically designed thread and needle are used, the internal os is stitched together like the mouth of a closed purse or pouch.
- Internal os is the junction of uterus and cervix.

Q. No h/o Abortions, no h/o Pre-Term labour, did USG at 18-24 wks. Cervical length < 25mm. Next Step?

Ans: Pt is HIGH RISK for Pre-Term Labour. So, give PROGESTERONE SUPPLIMENTATION

Q. If h/o Pre-Term Labour ; USG 18-20wks, Cervical length < 25mm. What next?

Ans: Cervical Cerclage + Prophylactic Progesterone Supplementation

Q. Diagnosis of cervical incompetence in Non-Pregnant state?

Ans: If No.8 HEGAR's dilator can be passed through Internal OS without resistance. done in PRE-MENSTRUAL phase.

iv. APLA SYNDROME

1 Clinical criteria + **1 Lab Criteria**

1 out of 4

1. ≥ 3 pregnancy loses at <10wks
2. ≥ 1 pregnancy loss beyond 10wk (>10wks), should be of a morphology Ⓝ fetus
3. Atleast 1 Pre-Term delivery <34wks 2° to UPI or SEVERE PRE-ECLAMPSIA
4. H/o Thrombosis

ANTIBODIES

1. LUPUS ANTI COAGULANT (LAC)
2. ANTI CARDIOLIPIN Ab (ACA)
3. ANTI β_2 GP1 Ab

Look for IgG or IgM Abs +ve in medium to high titre on 2 occassion 12wks apart

K/c/o APLA in Pregnancy : Rx - 1. HEPARIN (LMWH > Unfractionated Heparin)

2. LOW DOSE ASPIRIN

Both drugs continued throughout pregnancy.

HEPARIN is to be given only if there is h/o ABORTION or THROMBOSIS

DOC for APLA : WARFARIN

- **INFECTIONS** that donot cause **RECURRENT PREGNANCY LOSS** are

1. TORCH
2. SYPHILIS - • cause EARLY RPL ; doesnot cause RECURRENT EARLY RPL

• **KASSOWITZ LAW** - c̄ every Pregnancy POG of loss keep increasing

5 TYPES OF ABORTIONS : Based on PRESENTATION

- MISSED
- THREATENED
- INEVITABLE

PRESENTS c̄ AMENORRHOEA + PAIN + BLEEDING

- INCOMPLETE
- COMPLETE

PRESENTS c̄ AMENORRHOEA + PAIN + BLEEDING + h/o EXPULSION OF PRODUCTS

MISSED	THREATENED	INEVITABLE	INCOMPLETE	COMPLETE
CERVICAL OS : CLOSED	CLOSED	OPEN	OPEN • Pdts of conception in Cervical canal	CLOSED
UTERINE SIZE : < POG	Correspond to POG	Correspond to POG	< POG	< POG
USG done : Cardiac activity - ABSENT INDUCE ABORTION	Cardiac activity - PRESENT NO SPECIFIC Rx EMPIRICAL 1. Rest 2. Progesterone Supplementation 3. Avoid lifting weight 4. AVOID COITUS	Products will any how EXPELLED	MANAGEMENT • Complete the process of expulsion done by SUCTION and EVACUATION • Can present as SHOCK	MANAGEMENT On USG : CONFIRMATION showing EMPTY UTERUS.

INDUCED ABORTION

- MTP Act 20wks
- If POG : <12 - 1 Registered Medical Practitioner

  12-20wks - 2 Registered Medical Practitioners

- Done in
  1. RAPE VICTIM - Humanitarian
  2. GCA - Eugenic
  3. Medical / Surgical Ds - Medical
  4. Contraceptive Failure - Social

1st TM MTP

upto 7wk - OUTPATIENT MEDICAL ABORTION

- Tab. MISOPROSTOL
- Tab. MIFEPRISTONE

- On Day 1 : Tab. MIFEPRISTONE - 200mg

  Day 3 : Tab. MISOPROSTOL - 400mcg (ORAL / BUCCAL / S/L / VAGINAL)

  Day 15 : H/o, Examine pt. Bleeding happens after MISOPROSTOL

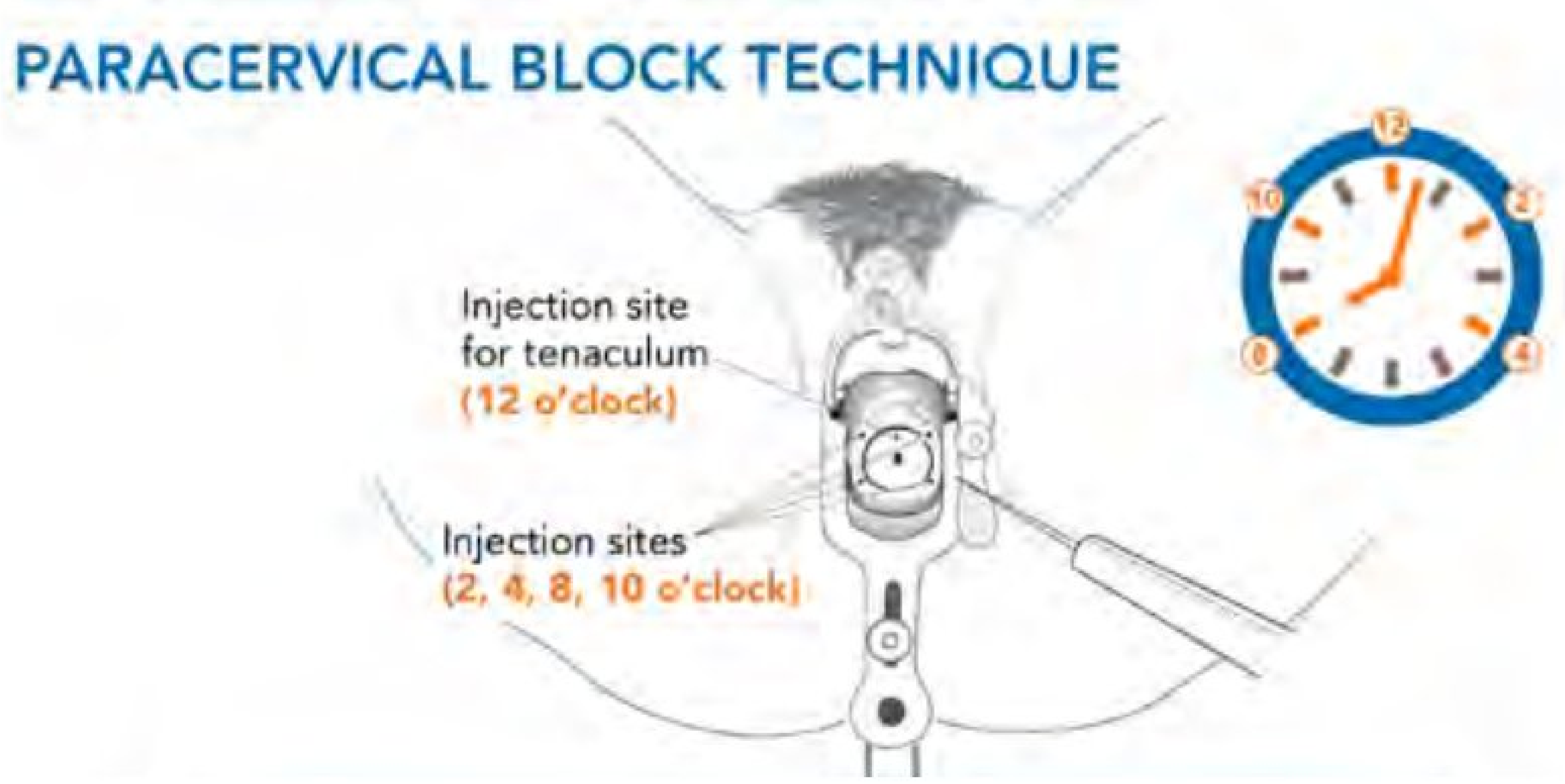
- USG BEFORE or AFTER the procedure is NOT MANDATORY

- **MOBIUS SYNDROME**: 6th / 7th Nr. Palsy d/t MISOPROSTOL

BEYOND 7-12wks - SUCTION and EVACUATION

- Dilatation Cervix cause pain ⟶ can be prevented by giving PARACERVICAL BLOCK
  - at 4 and 8 'O' Clock position or at 2 and 10

NOT at 3 and 9 'O' clock
because there is
Descending Cervical Artery -
Br. of Uterine artery


PARACERVICAL BLOCK TECHNIQUE
Injection site for tenaculum (12 o'clock)
Injection sites (2, 4, 8, 10 o'clock)
12
2
4
8
10

- Before using HEGAR's DILATOR - 1st use UTERINE SOUND - done to know position of the uterus
- If Perforation ≤ HEGAR's DILATOR ⟶ Monitor VITALS ⟶ Wait and Watch
- If Perforation during SUCTION EVACUATION ⟶ Do IMMEDIATE LAPAROSCOPY

END POINT OF SUCTION EVACUATION (Suction Pressure - 600mmHg)

1. ↓ Bleeding
2. AIR BUBBLES in CANULA
3. Gripping Sensation on CANULA
   ↓
   Check CURETTAGE (BLUNT)

MVA SYRINGE ⟶ Alternative to Suction and Evacuation
[BIVALVE SYRINGE]
- upto 12wks
- 60ml volume
- 660mmHg (∼ 600mmHg)

BEYOND 12 wks upto 20wks

I. MEDICAL METHODS

- In Patient Abortion - MC done

Drawback: Can remain INCOMPLETE
Then do SURGICAL

A. DRUGS - MC used

i) Prostaglandins : MC MISOPROSTOL
ii) GEMEPROST
iii) CARBOPROST

B. EXTRA-AMNIOTIC ETHACRIDINE

C. INTRA-AMNIOTIC HYPEROSMOLAR SALINE

D. OXYTOCIN

2. SURGICAL METHOD

Advantage : One Time Procedure

D and E [DILATATION and EVACUATION] done

Ovum forceps used. Suction Canula NOT USED

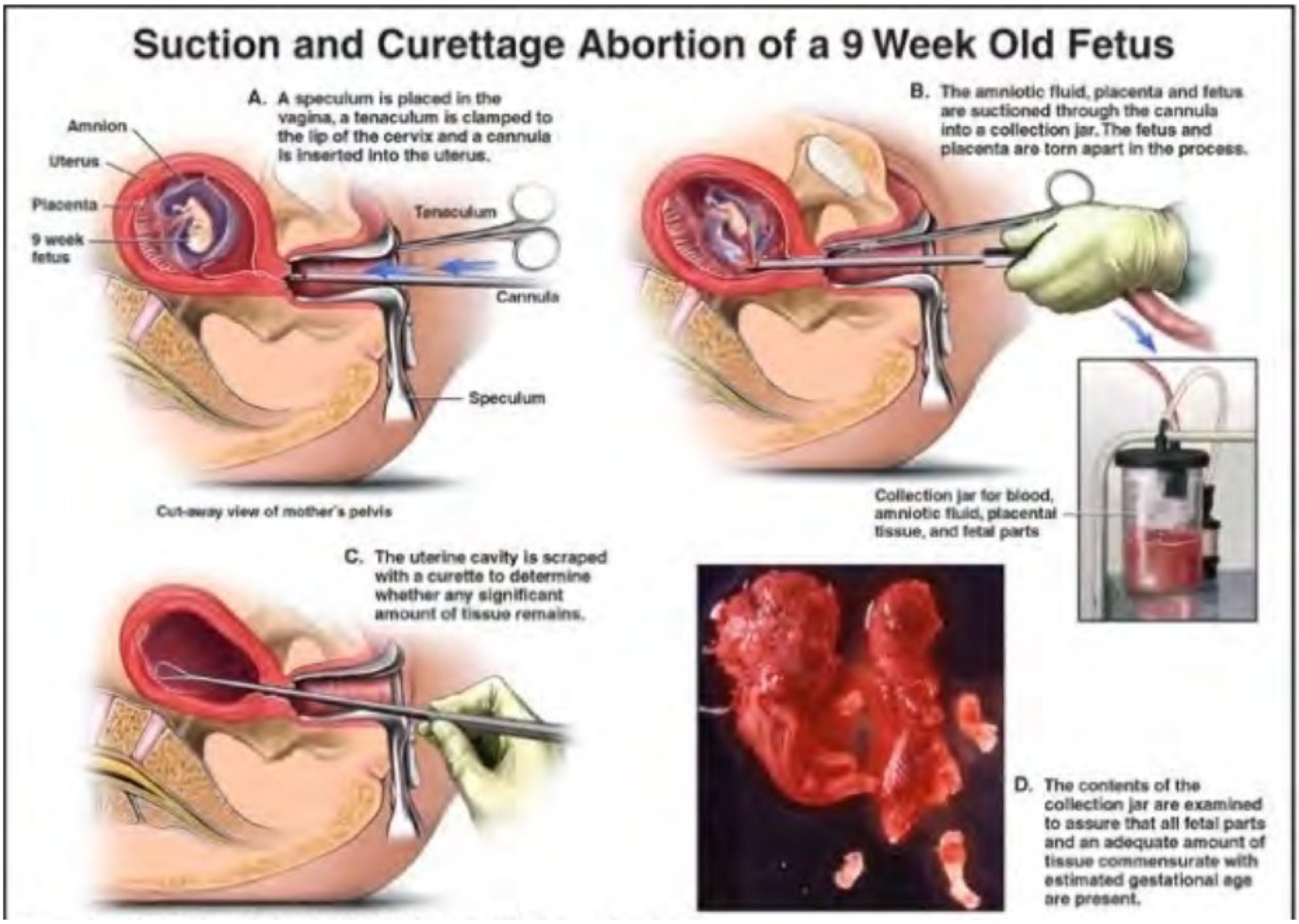
↓

Then do CHECK CURETTAGE [BLUNT]

Preg. Cervix - use Sponge holder

Non-Preg. Cervix - use Vulsellum

Manual Vacuum Aspiration (MVA Kit)



Suction and Curettage Abortion of a 9 Week Old Fetus

A. A speculum is placed in the vagina, a tenaculum is clamped to the lip of the cervix and a cannula is inserted into the uterus.

B. The amniotic fluid, placenta and fetus are suctioned through the cannula into a collection jar. The fetus and placenta are torn apart in the process.

C. The uterine cavity is scraped with a curette to determine whether any significant amount of tissue remains.

D. The contents of the collection jar are examined to assure that all fetal parts and an adequate amount of tissue commensurate with estimated gestational age are present.

Amnion
Uterus
Placenta
9 week fetus
Tenaculum
Cannula
Speculum
Cut-away view of mother's pelvis
Collection jar for blood, amniotic fluid, placental tissue, and fetal parts

ECTOPIC PREGNANCY

- 2% of all Pregnancy ; MORTALITY : >10%

- Pregnancy Implants outside Uterine cavity

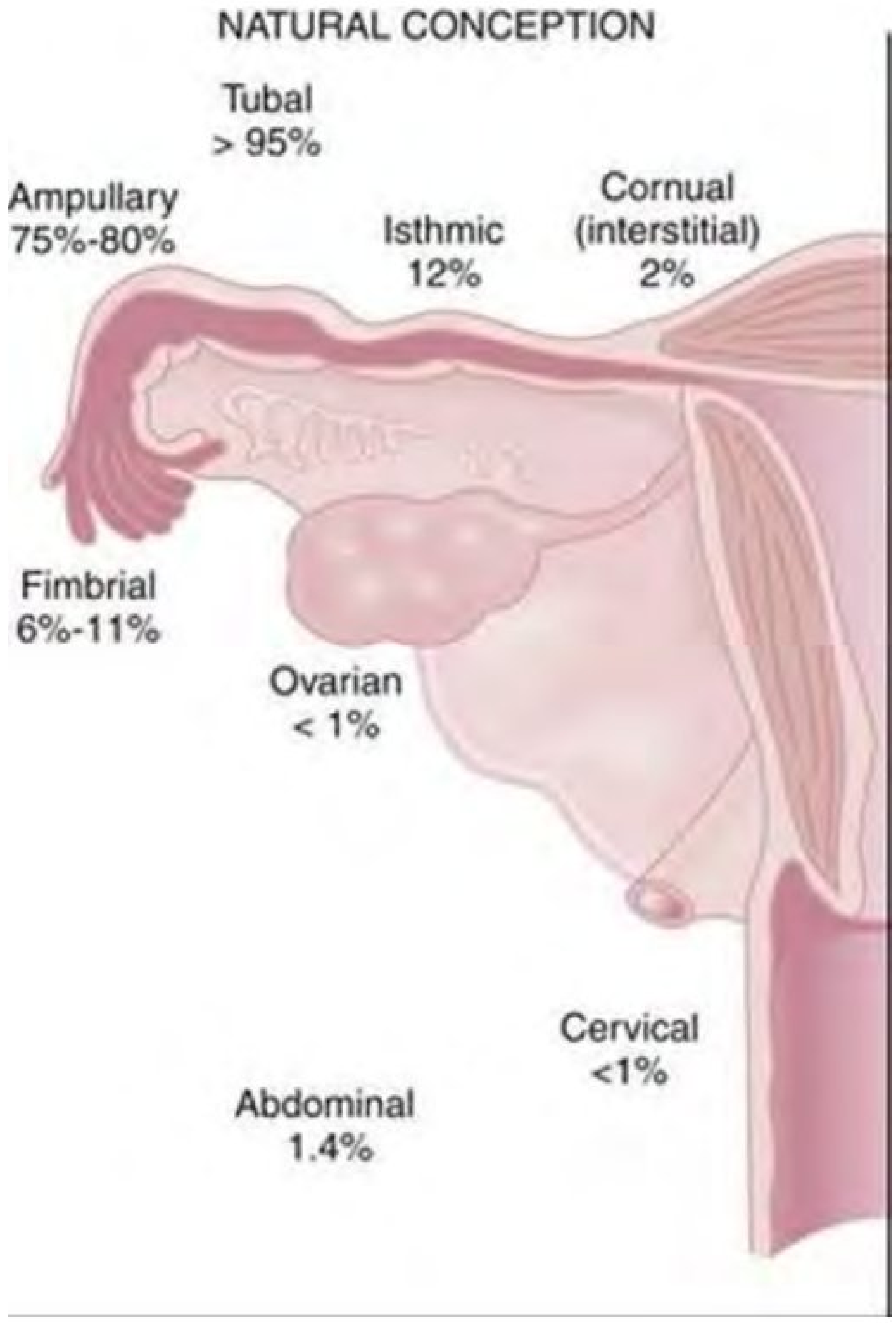
(MC) Site - Fallopian Tube

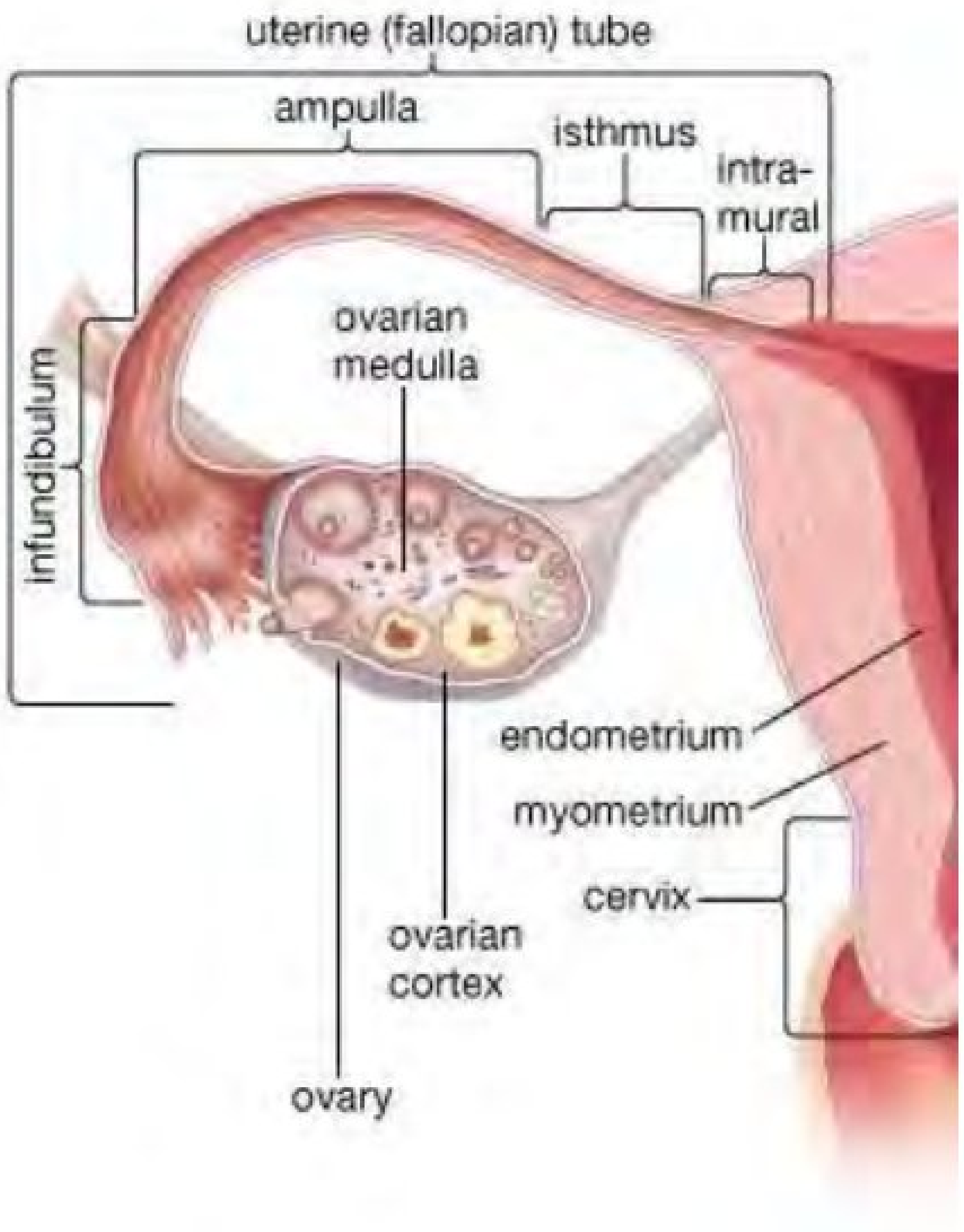
(LC) site - C.S Scar Pregnancy > Cervical Pregnancy > Abdominal ↳ 1%

C.S Scar Pregnancy, Cervical Pregnancy: <1%

- **Parts of FT**

1. INTRAMURAL / INTERSTITIAL - Narrowest : 0.7mm dia
2. ISTHMUS
3. AMPULLA → Widest part : 6mm dia
4. INFUNDIBULUM





In Tubal Pregnancy - ZYGOTE Implants in Muscular layer

1. Site of Fertilization : AMPULLA (MC) site
   because it has Max^{m} no. of PLICAE (mucosal folds)

2nd (MC) site of Ectopic : ISTHMUS

(LC) site of Ectopic : INTRAMURAL / INTERSTITIAL

- Sudden narrowing at ISTHMUS acts like Physiological Sphincter
- INTRAMURAL / INTERSTITIAL part acts like ANATOMICAL Sphincter because Smooth muscles are arranged in a circular manner
- Main Reason for Transport of Conceptus : TUBAL PERISTALSIS
- Lining Epithelium of FT → Single Layer Ciliated Columnar Epithelium

Lining Epithelium - 3 Secretory cells

1. SECRETORY CELLS - Nutrition
2. CILIATED CELLS - Movement
3. PEG CELLS - Resting Cells → Converted to SECRETORY CELLS

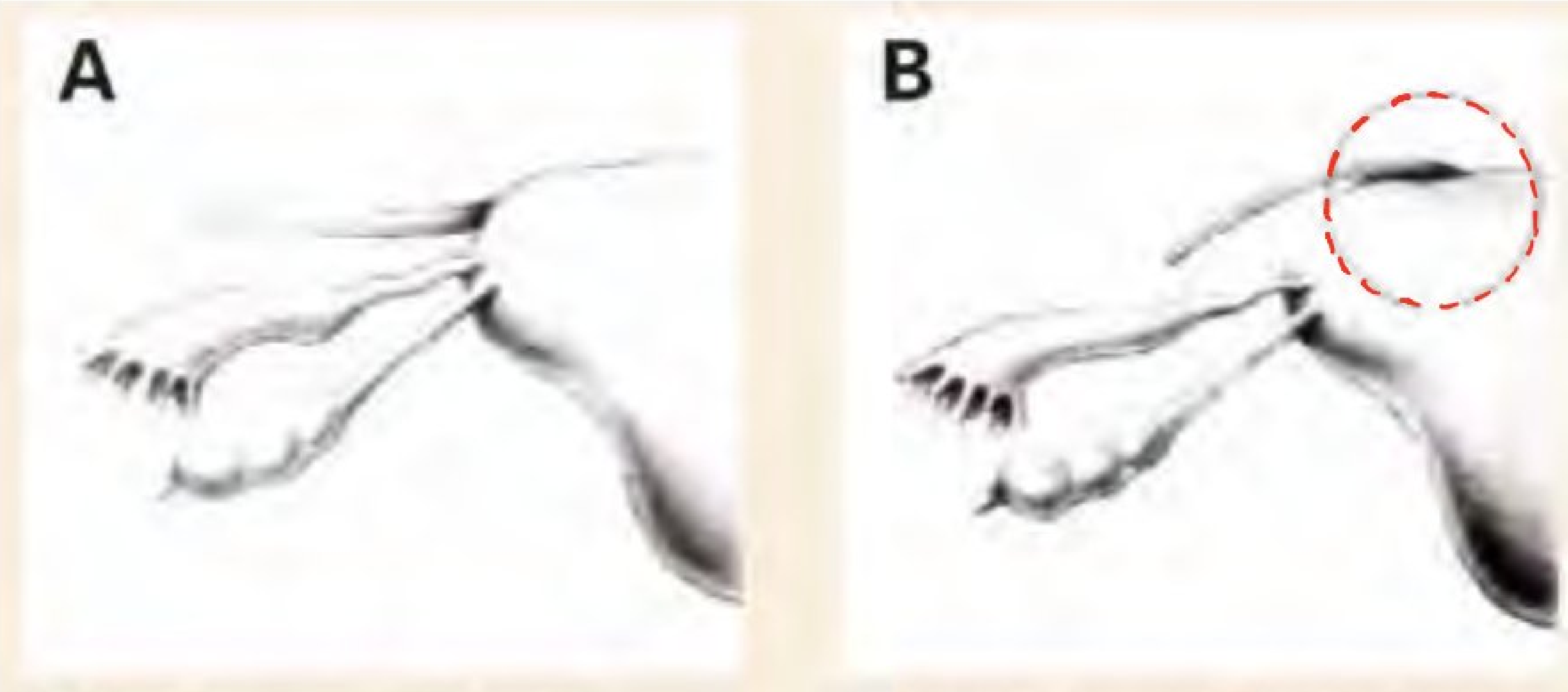
- Anything that REDUCES Tubal mobility will be Risk Factor of ECTOPIC

  Eg: PID / Tubal Sx / Progesterone

- Site for TUBAL LIGATION : ISTHMUS

  ↳ (MC) Reason for FAILURE is ligating the wrong structure

- ADNEXA = FALLOPIAN TUBE + OVARY [BABCOCK FORCEPS is used to hold all luminal structure and is ATRAUMATIC]



A) Angular pregnancy with a cornual swelling that does not distort the anatomical relationships between the round or ovarian ligaments and the fallopian tube.

B) Cornual or interstitial pregnancy, here marked by a cornual swelling that displaces the round ligament anteriorly as it erodes through the anterior wall of the tube.

RISK FACTORS FOR ECTOPIC

1. Highest Risk - H/o Prev. Ectopic : 1 prev. → 15%
   2 prev. → 30%
2. H/o TUBAL Sx - 2^{nd} Highest Risk
3. (MC) R/F → PID (Chlamydia Trachomatis)
4. CERVICITIS
5. Multiple Sexual Partners
6. Smoking
7. H/o INFERTILITY
8. Ceasarean Section
9. CONTRACEPTION and ECTOPIC

CONTRACEPTION

CONTRACEPTION → prevents Intrauterine but NOT Extra uterine Pregnancy

↳ AGENTS → ↓ the ABSOLUTE RISK of ECTOPIC

↳ Some Contraceptive Agents which will ↑ Relative Risk

Currently using these methods - If fails → ECTOPIC PREGNANCY

- Methods are : TUBAL STERILIZATION or LIGATION > IUD > POP

$\underbrace{\text{PROGESTASERT} > \text{MIRENA}}_{\text{Progesterone containing IUD's}} > \text{CuT}$

MIRENA

- Releases LNG 20 mcg /day
- Approved for 5yrs
- F/R - 0·1 - 0·2 %
- Total LNG - 52mcg

PROGESTASERT

- Has Higher Failure Rate : So WITHDRAWN
- Releases 65 mcg / day

OUTCOMES IF ECTOPIC

1. At AMPULLA → TUBAL ABORTION (MC)
   ↳ 2nd (MC) Tubal Rupture ; Ruptures around 8wks of Pregnancy
2. ISTHMIC PREGNANCY (MC) Outcome - Rupture at 6wks
   ↳ (MC) Ectopic to Rupture
3. INTRAMURAL / INTERSTITIAL PREGNANCY - Rupture at 12wks
   - Amount of Blood loss more when compared c̄ others → MORE LIFE THREATENING (Highest Mortality)

(LC) site for Rupture - FIMBRIAL END

- In Ruptured Ectopic - Temp Ⓝ/↓
- In Salpingitis - Temp ↑

CLINICAL PRESENTATION

TRIAD : PAIN + AMENORRHOEA + BLEEDING
↓
s/i only 50% Pt

(MC) / Most consistent : PAIN
- → Ruptured Ectopic → HEMOPERITONEUM
  ↓
  PERITONITIS
- → if UNRUPTURED d/t TUBAL STRETCHING
- Nerve fibres responsible is T_{11}, T_{12}, L_1

Other features :

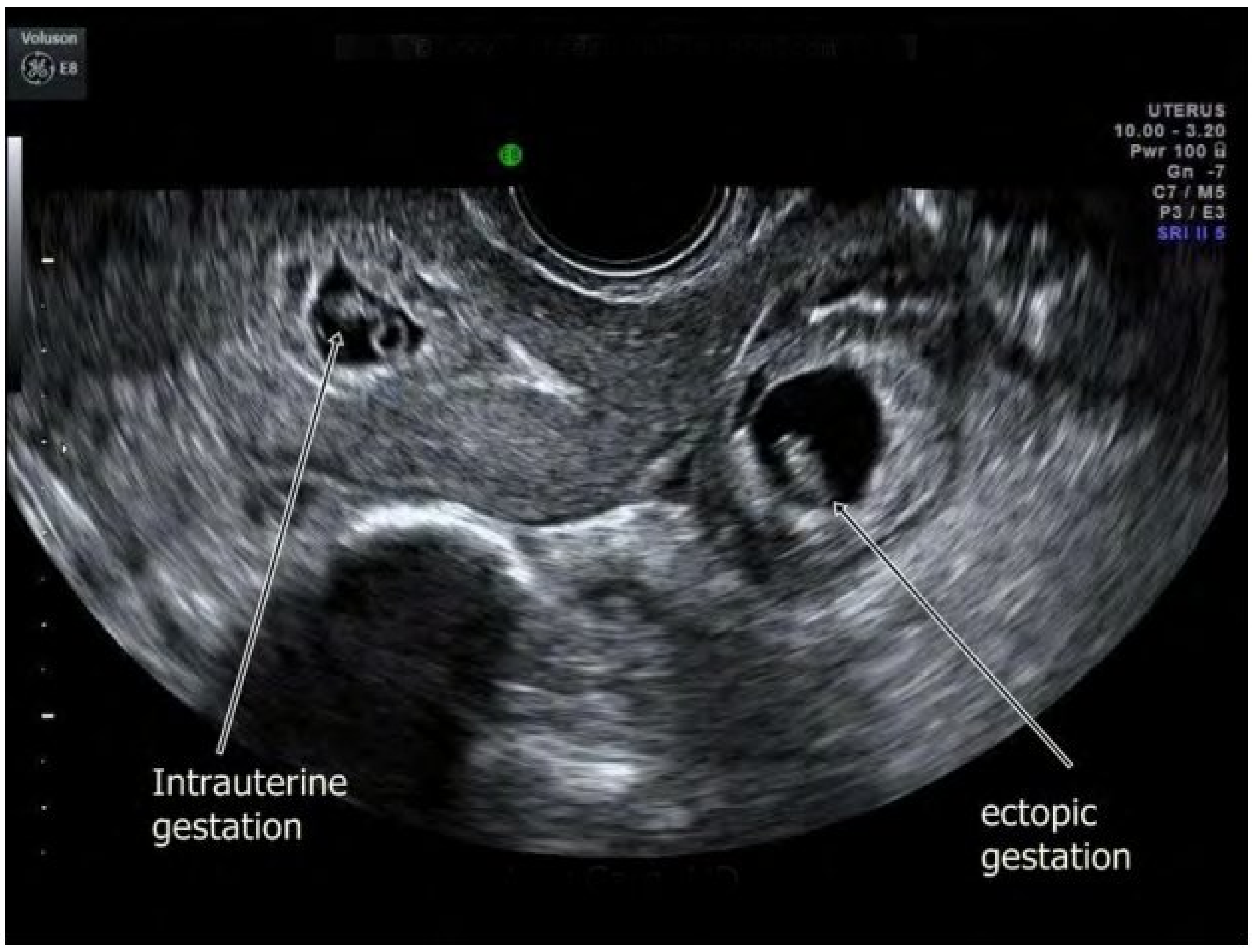
- Nausea, Vomiting
- Postural Hypotension
- Ⓡ Shoulder tip pain - REFFERED PAIN → Irritation of Diaphragm
- PR ± TACHYCARDIA
- BP ± HYPOTENSION
- **On P/A** - Tenderness in Lower Abdomen
  ± GUARDING / RIGIDITY } LOCALISING SIGNS of PERITONITIS, RUPTURED ECTOPIC
- **On P/V** - Uterus Soft / Enlarged (Size of Uterus LESS than POG)
  - CERVICAL MOTION TENDERNESS → s/i
    - Ruptured / Rupturing Tubal Pregnancies - 75%
    - Salpingitis → PID
  - ADNEXAL TENDERNESS
  - ADNEXAL MASS
- 1st Step : (MC) HCG UPT : 99% UPT ⊕ve

IOC for ECTOPIC - TVS USG → EXTRA-UTERINE G.sac c̄ YOLK SAC / EMBRYO

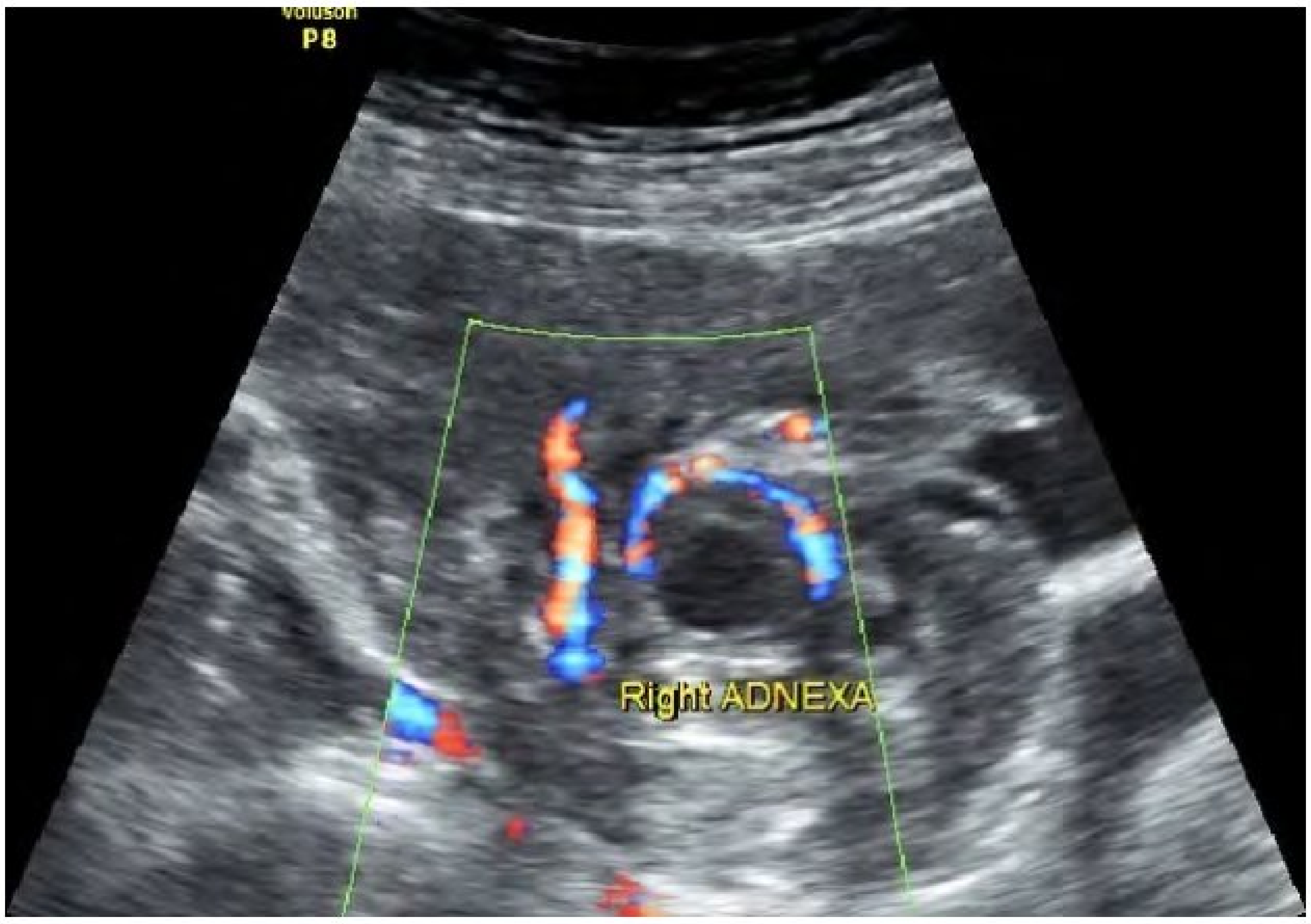
FINDINGS on USG s/o ECTOPIC

- Empty uterus c̄ Complex Adnexal mass (some FLUID + some WHITE areas)
- CYSTIC MASS in EARLY PREGNANCY

  in OVARY → CORPUS LUTEUM



On DOPPLER : "RING OF FIRE" → S/o HIGH VASCULARITY



if S/o ECTOPIC → pt undergo → SERIAL β-HCG Assessment : DAY 1 - 1st Sample

↓ After 48hrs

DAY 3 - 2nd Sample [SLOW RISE < 55%] → Begins Rx of ECTOPIC

if D1 value itself ≥ 2000 [CRITICAL Value]
+
USG Features S/o ECTOPIC
} Rx for ECTOPIC

Q. USG if No IntraUterine Preg. B/L Adnexa - Ⓝ ; HCG ⊕. What Next?

Ans : It could be
1. ECTOPIC
2. NORMAL
3. ABORTION
} Do Serial HCG → D1 → D3

D3 → SLOW RISE - S/o ECTOPIC
D3 → DOUBLING - S/o INTRA UTERINE
} Repeat USG

D3 → ↓↓ - S/o ABORTION

3. Best Investigation for ECTOPIC : DIAGNOSTIC LAPAROSCOPY

4. S.Progesterone level ≥ 25ng - VIABLE INTRA-UTERINE Pregnancy

< 5ng - S/o ECTOPIC, ABORTION

5. CULDOCENTESIS

- Posterior wall of vagina longer than Anterior by 2cm
- Posterior Fornix deeper than Anterior fornix
- Recto uterine pouch / Pouch of DOUGLAS / Cul-de-sac → Most dependent part of Female pelvis

Aspirate from POUCH OF DOUGLAS

↳ BLOOD → **HEMOPERITONEUM** : s/o Ruptured Ectopic

- **On USG** : >100ml - SIGNIFICANT
- COLPOTOMY - to drain a Pelvic abscess

TREATMENT OF ECTOPIC

1. Medical Mx
2. Surgical Mx
3. Expectant Mx

1. MEDICAL MANAGEMENT

- gives option of preserving the fertility
- Requirements for Medical Mx → fails in 5-10 % cases

① Pt. stable

② unruptured

③ Cardiac activity - ABSENT

④ G.sac size - sac size < 3cm - 3.5cm , Cardiac activity ⊕

- sac size < 4cm - if Cardiac activity ABSENT

⑤ HCG < 5000 IU

⑥ Dead fetus

- **METHOTREXATE : DOC - 1mg/kg i.m**

D_1 - Inj. METHOTREXATE

D_4 - S.HCG

D_7 - S.HCG → if ↓ (Fall by MINIMUM of 15%) → Successful Med Mx : βHCG disappears from plasma b/w 14 - 21 days.

↳ if falls by < 15% → NOT SUCCESSFULL

↓

So, Repeat Inj. METHOTREXATE → becomes D_1 → D_4 - S.HCG

D_7 - S.HCG

- **Medical Mx failed if Max^m Total dose of 3 METHOTREXATE INJECTION**
- " SINGLE DOSE THERAPY "

2. SURGICAL MANAGEMENT

- **INDICATIONS OF SURGERY**

1. Unstable
2. Ruptured Ectopic
3. Cardiac activity ⊕ in G.sac
4. Criteria for sac size NOT met
5. HCG > 5000 IU
6. Family is Complete
7. Medical Rx FAILS

- LAPAROSCOPY > LAPAROTOMY
- UNSTABLE VITALS → do LAPAROTOMY
- SALPINGOSTOMY > SALPINGECTOMY → Indications :
  1. Ruptured Ectopic
  2. Family Complete
  3. Sac Size > 5cm
  4. If hemostasis can't be maintained

  SALPINGOSTOMY ↓ done to preserve fertility

ABORTION :
- follow up Serial βHCG till it returns to Ⓝ, i.e ∼ <5 IU
- Monitor VITALS

Q. Pt. comes ≤ 6wks Amenorrhoea and spotting. UPT ⊕ve, HCG = 2500 IU. USG shows EMPTY SAC.
What Next ?

Ans: Do Serial βHCG

Q. Pt ≤ 6wks Amenorrhoea, pain + Bleeding BP = 80/60 mm Hg, PR - 126/min, P/A examⁿ - Guarding
Next Step ?
Ans: Laparotomy

Q. Pt. married for 5yrs, 1° infertility, now conceived, presents as ruptured ectopic. BP - 106/80mmHg
PR - 104/min. What Next ?
Ans: Laparoscopy + Salpingectomy

CRITERIAS : ECTOPIC

CERVICAL
1. PAALMAN'S
2. RUBIN's

ABDOMEN
SLUDDIFORD ECTOPIC
- Survive Longest - 2nd/3rd TM
- Placenta attached to VITAL ORGANS → Obstetric Emergency ↓ Mx: Emergency Laparotomy
  ↓
  deliver Baby but donot Remove Placenta
  ↓
  Placenta left in-situ
  Left for AUTODIGESTION

SPIELBERG CRITERIA

HETEROTOPIC PREGNANCY

- 2 pregnancy simultaneously present at different sites of Implantation
- Ⓜⓒ cause : ART ; Rare but ↑ Incidence d/t IVF ; Ⓜⓒ Type → 1 Intrauterine + 1 Fallopian Tube
- Medical Mx : C/I ; Surgical Mx : Missed and present late → Rupture
  → Big Sac Size
- Rx OC : Laparoscopic Salpingectomy

GESTATIONAL TROPHOBLASTIC DISEASE

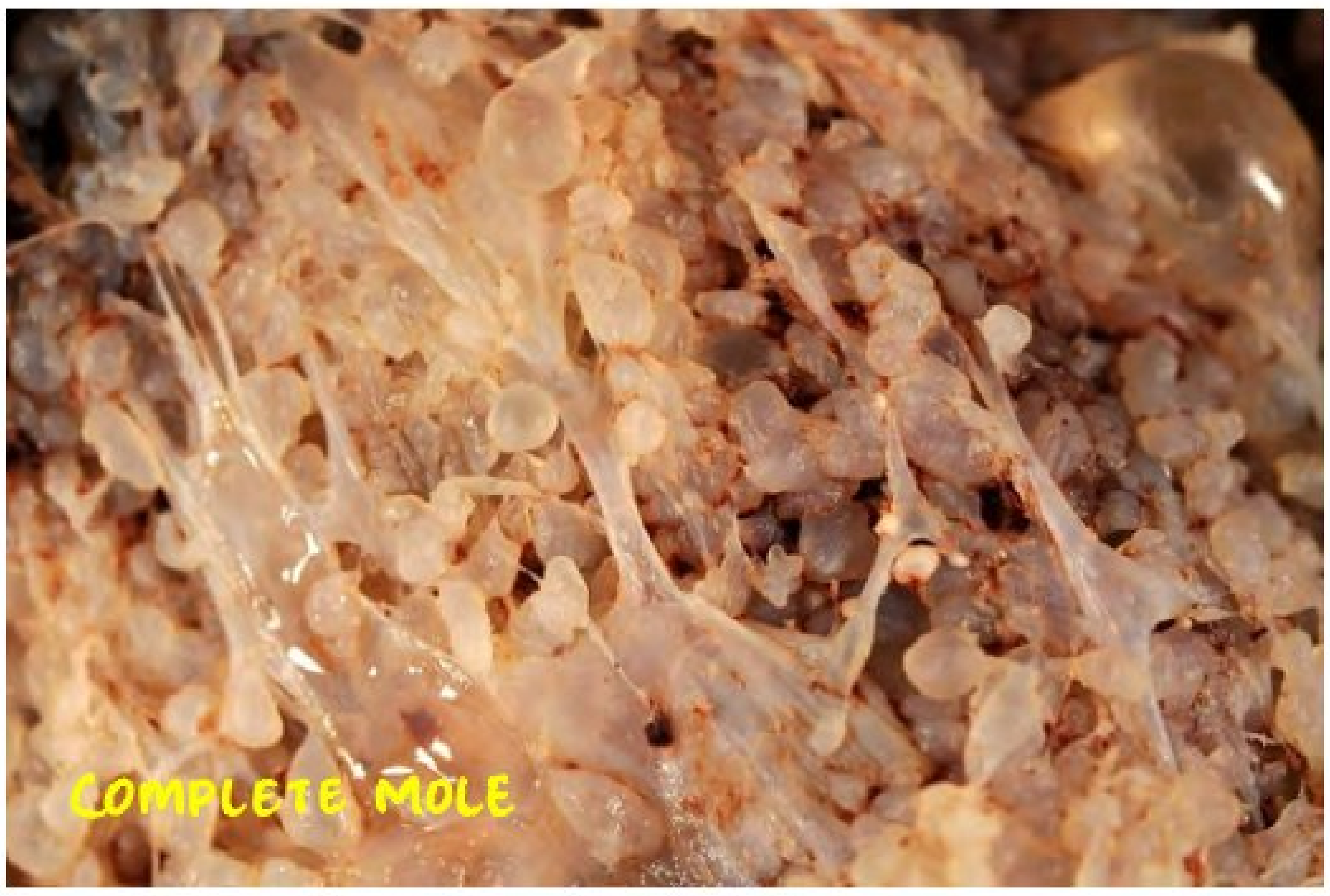
1. NEOPLASTIC 2. NON-NEOPLASTIC

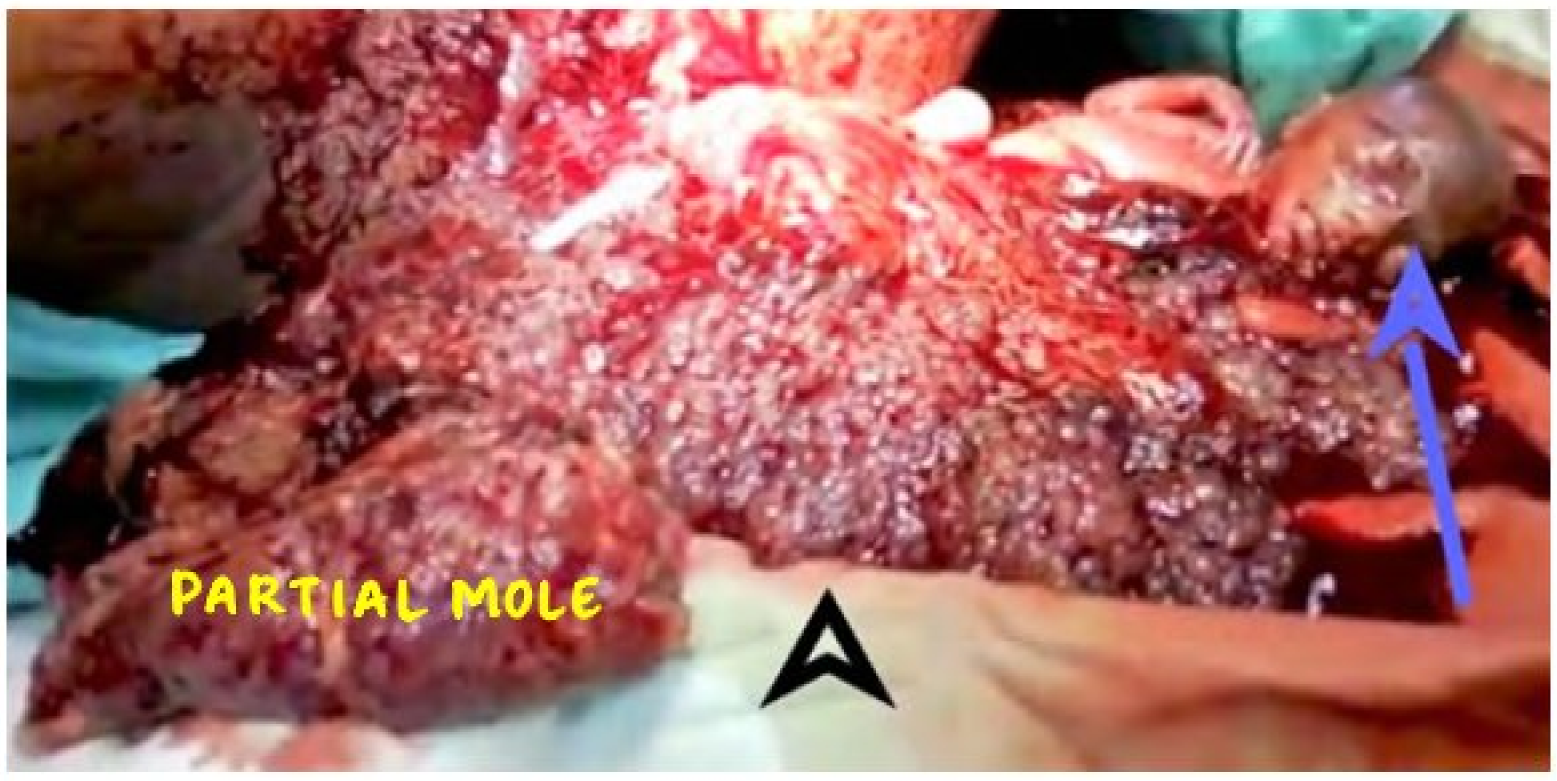
NON-NEOPLASTIC	NEOPLASTIC	
1. PARTIAL MOLE	1. INVASIVE Mole	3. Placental site Trophoblastic Tumor (PSTT)
2. COMPLETE MOLE	2. CHORIOCARCINOMA	4. EPITHELOID Trophoblastic Tumor (ETT)

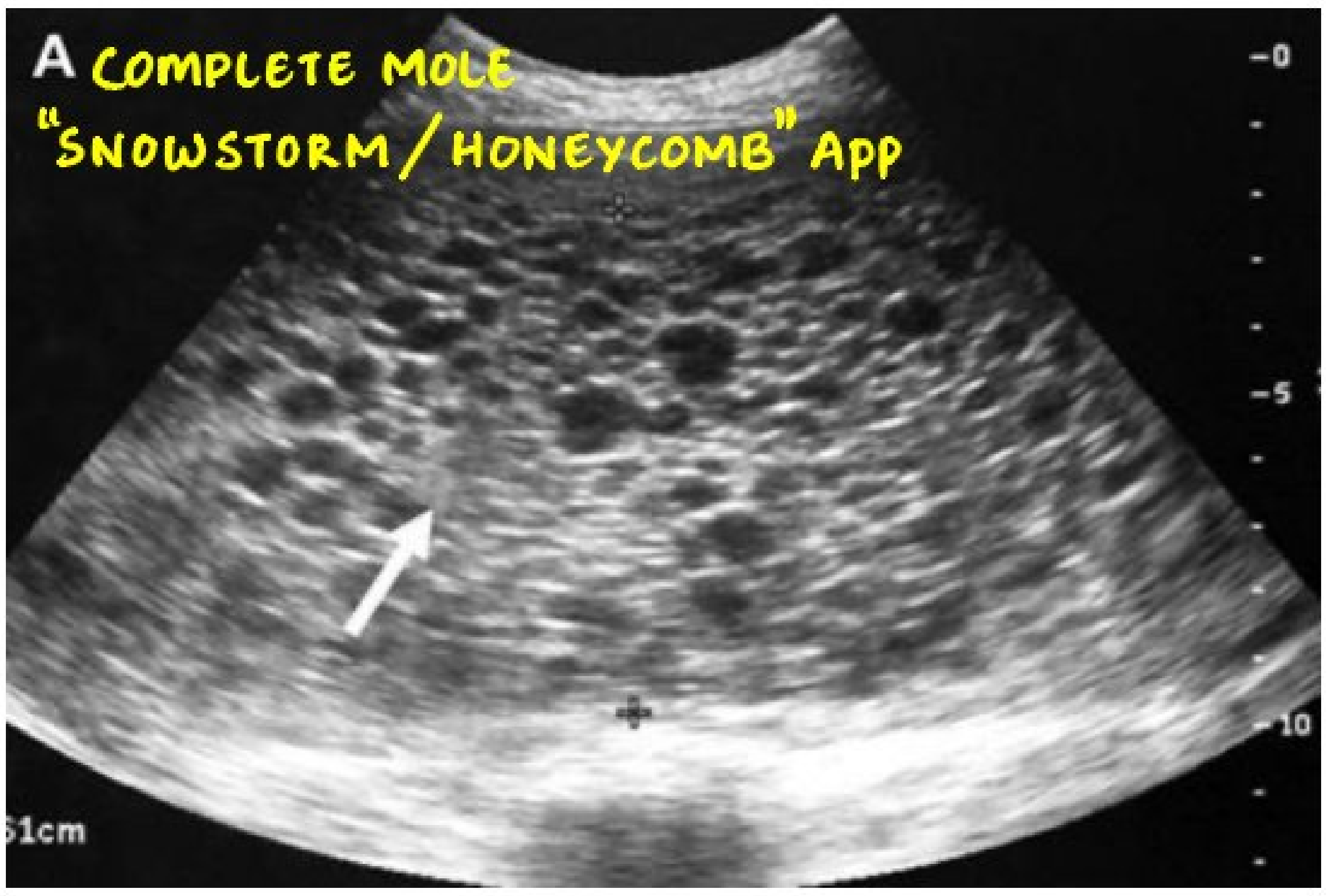
Complete and Partial Moles are usually d/o CHORION

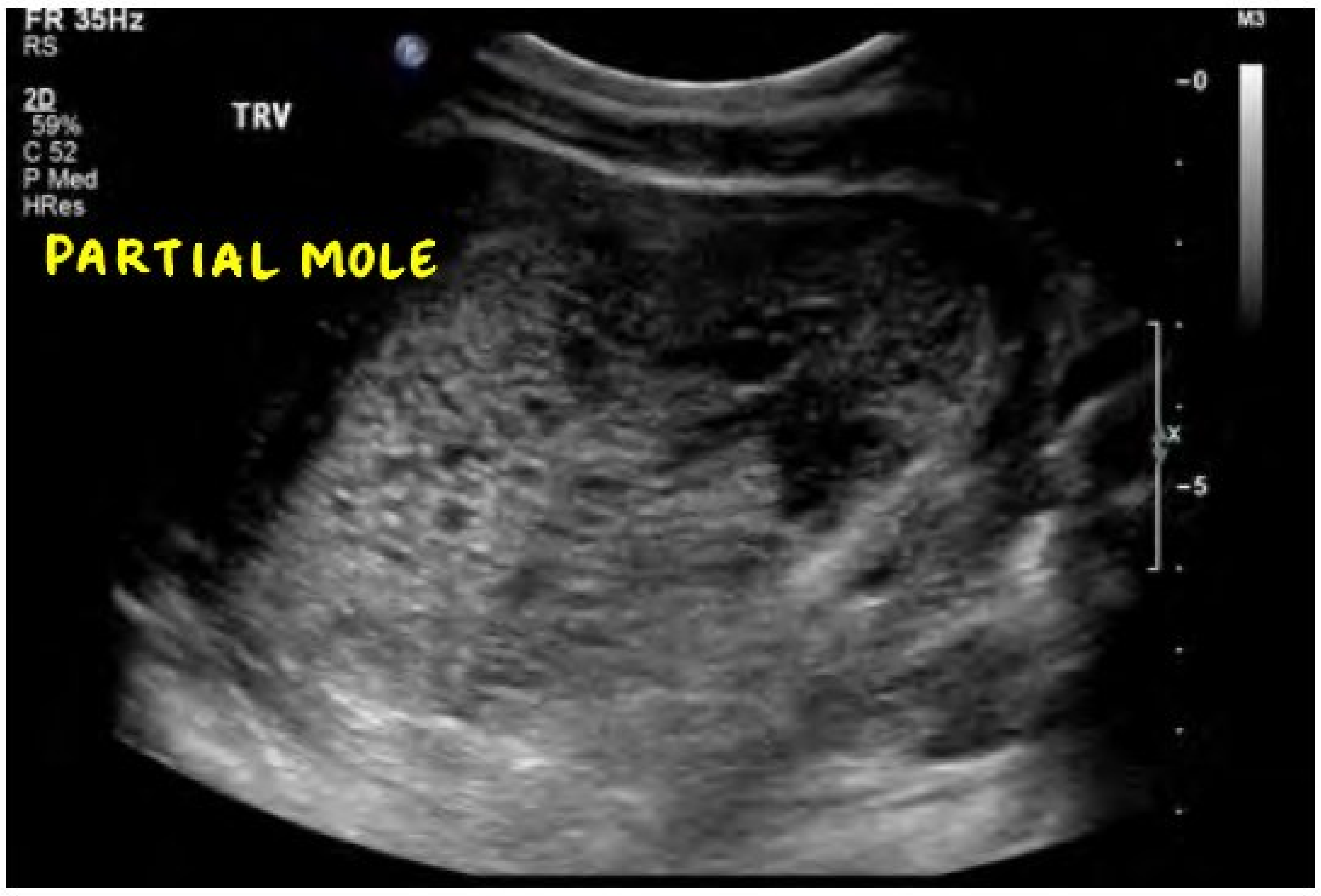
COMPLETE MOLE	PARTIAL MOLE
MC	LC
Diploid	TRIPLOID
Monospermic	Dispermic
All genetic material is PATERNAL	Extra set from Paternal Side
Genetic material of ovum degenerates this is fertilized by sperm and Paternal genetic material DUPLICATE	Ovum is fertilized by 2 sperms
MC - 46XX (80%) 46XY (20%)	90% TRIPLOID - 69XXY 10% TETRAPLOID - (3 sperms)
20% Dispermic	Abnormal Conceptus
On HPE [GOLD Std for Dx Confirmation]	
1. Extensive Trophoblastic Proliferation	1. Less marked
2. Complete Hydropic change of Villi	2. Focal Hydropic change
3. Avascular Villi	3. Vascular villi
4. No Fetal tissue	4. Fetal tissue - Present
5. No Trophoblastic Scalloping	5. Trophoblastic Scalloping - Present
6. No Stromal Inclusions	6. Stromal Inclusions - Present
	7. P57 Staining - Present

7. P57 Staining ⊖		
	CLINICAL PRESENTATION	
• Bleeding PV, Dark (Prune juice) • h/o passage of grape like vesicles	MC	• Bleeding PV
• Uterine size > POG	O/E	Uterine size ≤ POG
• very High (can be even $>10^5$)	HCG	Higher than expected (Not very High)
• No Fetus	USG IOC for MOLAR Pregnancy	• Fetus ⊕ → Multiple GCA
• Fluid filled vesicles s/a BLACK		• TRIPLOID → Die
• "SNOWSTORM / HONEYCOMB" App		⊗
• In Adnexa : Theca Lutein Cyst (cyst in the ovary)		⊗
• ↑ HCG (α Subunit) ~ LH - stimulate ovary ~ TSH		• P.moles - misdiagnosed on USG as MISSED ABORTION









Molar pregnancy (MC) Diagnosed in 1st TM and (MC) Presents in 2nd TM as Bleeding PV

Other Clinical presentations seen only in COMPLETE MOLE

1. Hyperemesis gravidarum
2. Early Onset Pre-Eclampsia
3. Thyroid storm
4. Pulmonary Embolism → d/t Small vesicles

Risk

C. mole → GTN : 15-20%

C. mole → Choriocarcinoma : 4%

P. mole → GTN : (3-5%)

P. mole → Choriocarcinoma : < 1%

MANAGEMENT

- TOC for Molar Pregnancy – SUCTION EVACUATION
- $\geq$ 40yrs C.mole and family complete – HYSTERECTOMY
- After Suction Evacuation – THECA LUTEIN Cyst regress c̄in 2-4 months
- During Suction Evacuation : There is Risk for
  1. THYROID STORM
  2. PULMONARY EMBOLISM – Highest

  We should start Suction 1st then Oxytocin
- Check curettage done : SHARP Curettage
- After Suction Evacuation : S. HCG checked weekly – till we get 3 consecutive values – Ⓝ

  ↓ after this monthly HCG
- Total Duration of Follow up / Surveillance after Molar = 6 months

  ↳ Advice is to : NOT to conceive

  [Average Time by which HCG returns to Ⓝ = 9 wks]

HIGH RISK for GTN

1. $\geq$ 40 yrs of age
2. Uterine size > POG
3. HCG > 10^5
4. B/L Large (6cm) Theca Lutein cyst.

- Contraception of choice is OCP's
  BARRIER's can be used [But has HIGH FAILURE Rate]
- Avoid - IUD → S/E Bleeding

- For those women at HIGH RISK → Prophylactic Chemo Rx → METHOTREXATE or ACTINOMYCIN-D

GTN - NEOPLASIA

1. Invasive Mole - (MC) GTN
2. Choriocarcinoma
3. PSTT } Rare
4. ETT }

- GTN (Neoplasia) can occur after Non-Molar pregnancy but uncommon
  ↓
  In this case (MC) GTN : CHORIOCARCINOMA

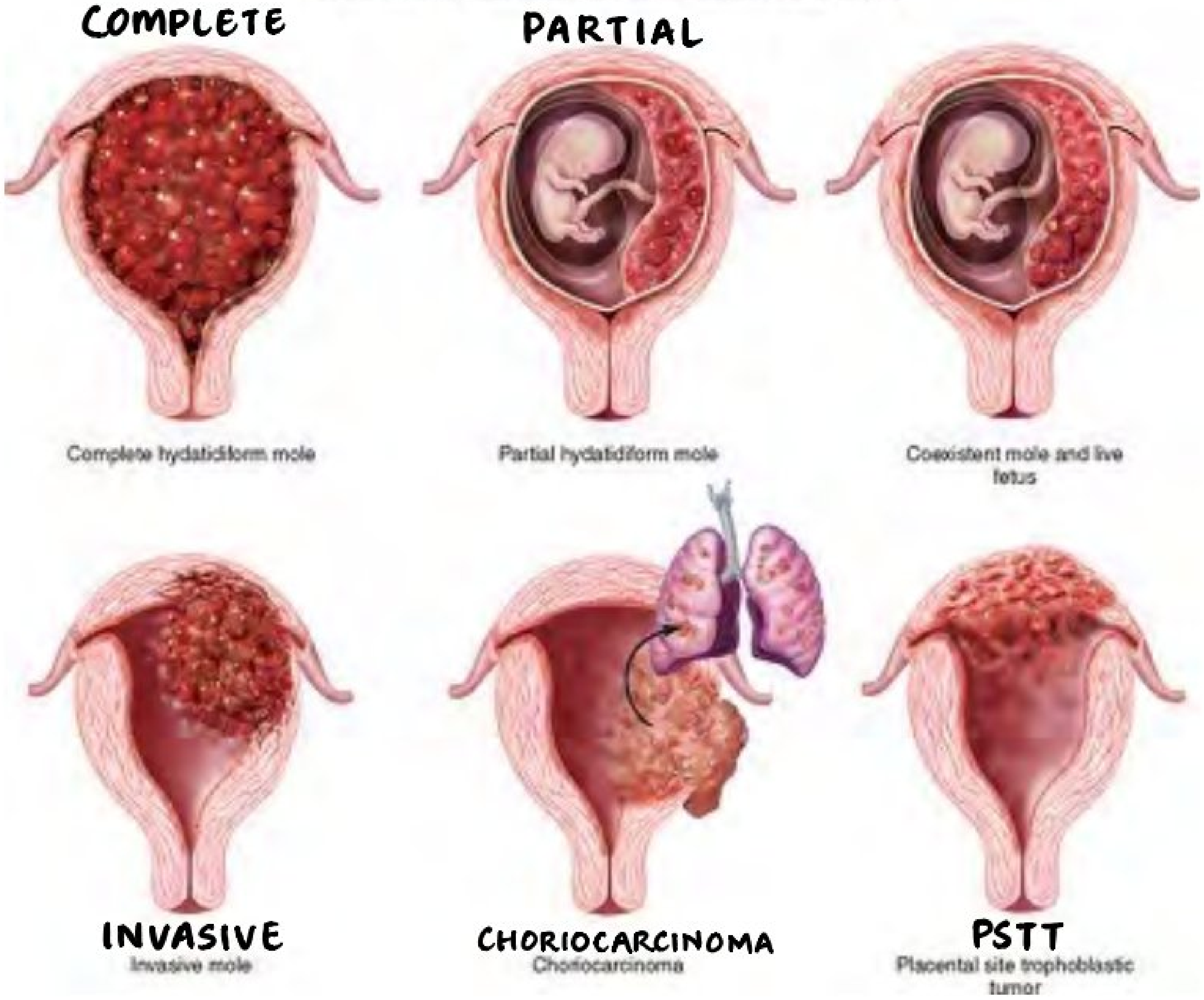
CRITERIA FOR Dx OF GTN

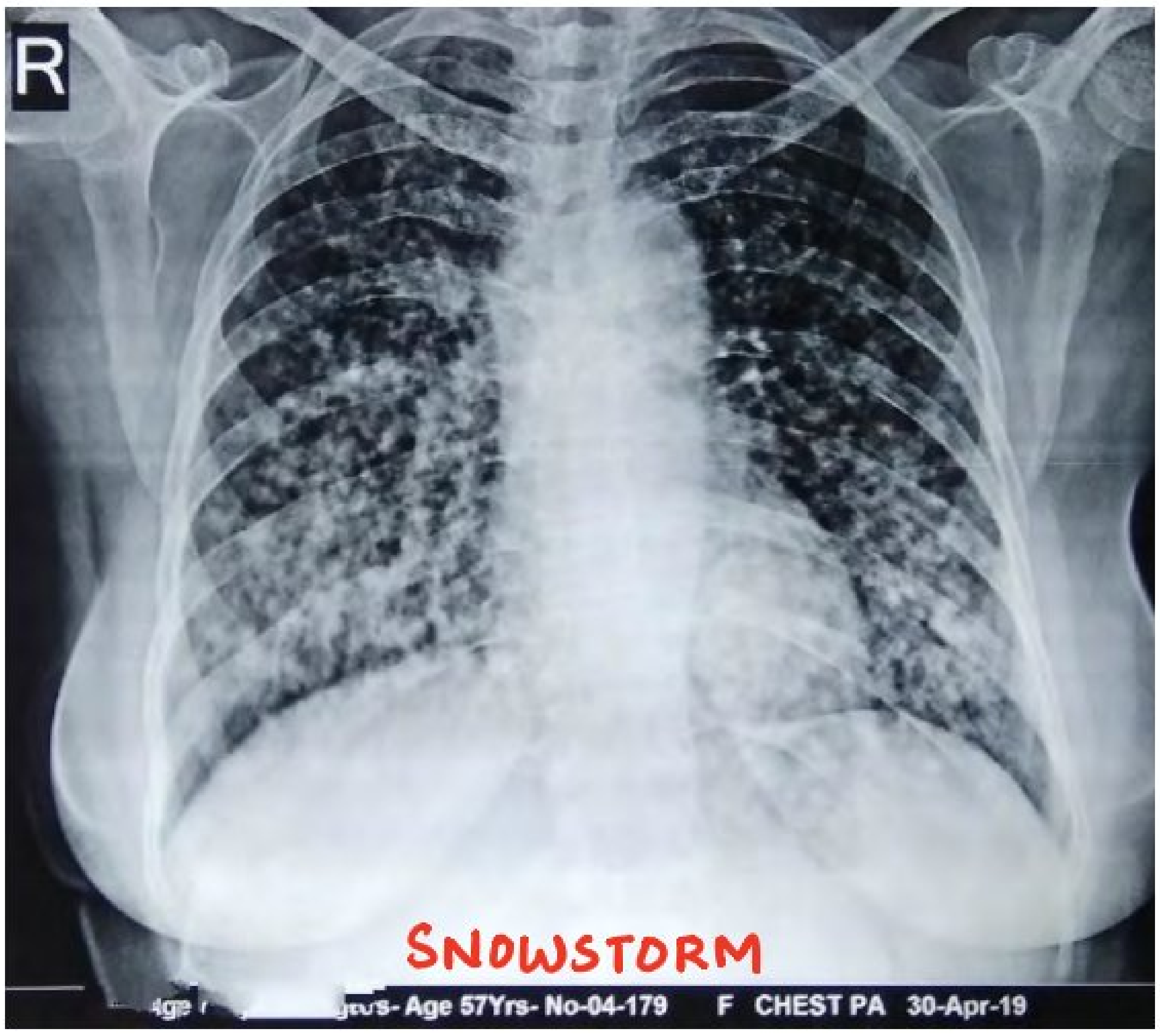
1. 4 consecutive values of HCG (D_1, D_4, D_7, D_{21}) (±10%) → shows PLATEAU
   ↳ S/i INVASIVE MOLE
2. 3 consecutive values of HCG (D_1, D_4, D_7) - shows a RISE (>10%) → S/i CHORIOCARCINOMA

CLINICAL PRESENTATION

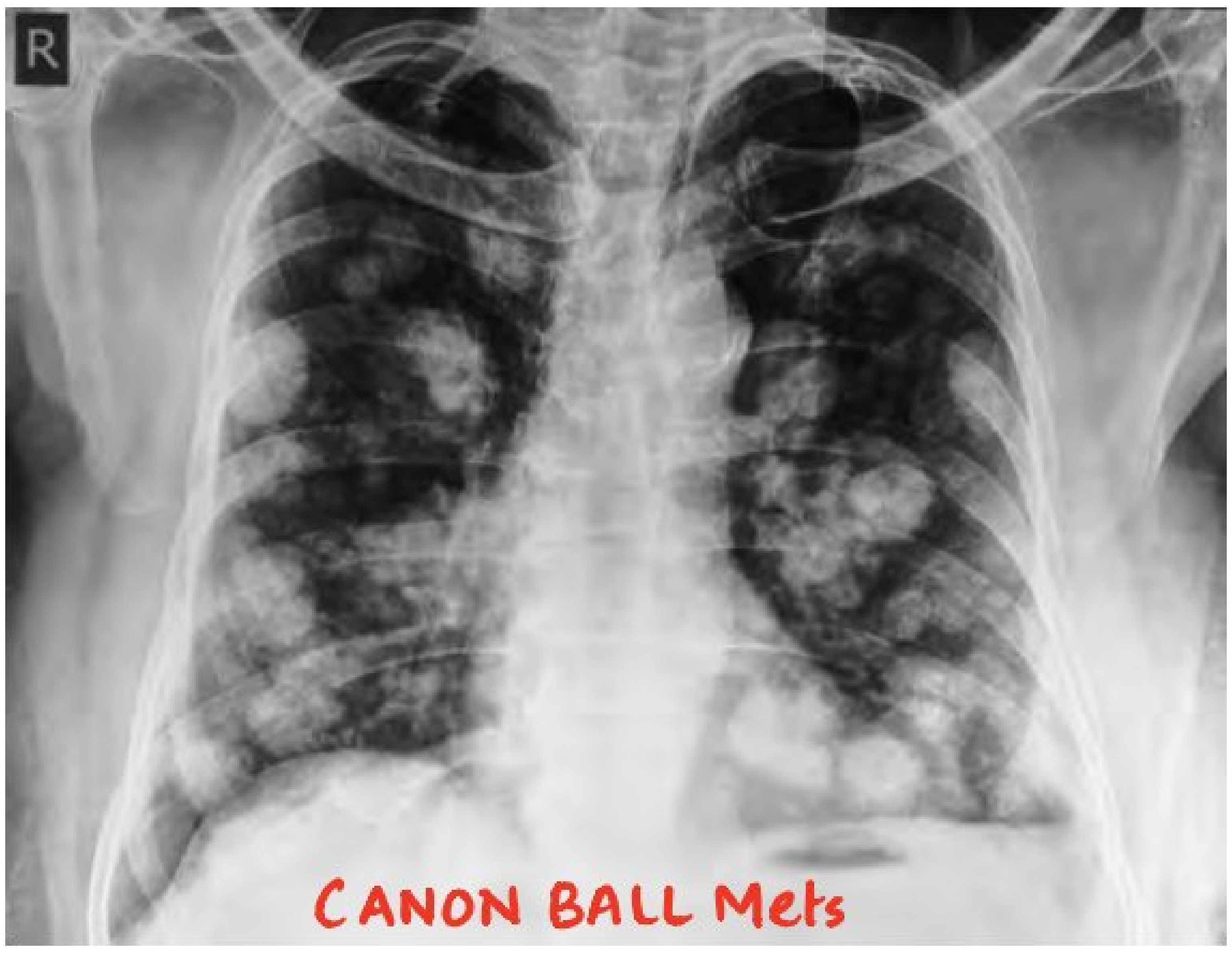
1. **Persistent bleeding** even after Suction and Evacuation
2. **Uterine subinvolution**
3. **Persistent Theca Lutein Cyst**
   ↳ pd HCG ↑; Choriocarcinoma
4. **SHOCK** - (Invasive Mole)
5. **METASTASIS** (Choriocarcinoma)
   ↓
   (MC) Site : Lungs > Vagina > Liver / Brain
   ↓
   On CXR: CANON BALL Mets - (MC)
   SNOWSTORM - 2nd (MC)
   in Vagina - Bluish Suburethral Nodule

SPECTRUM OF GESTATIONAL TROPHOBLASTIC NEOPLASIA

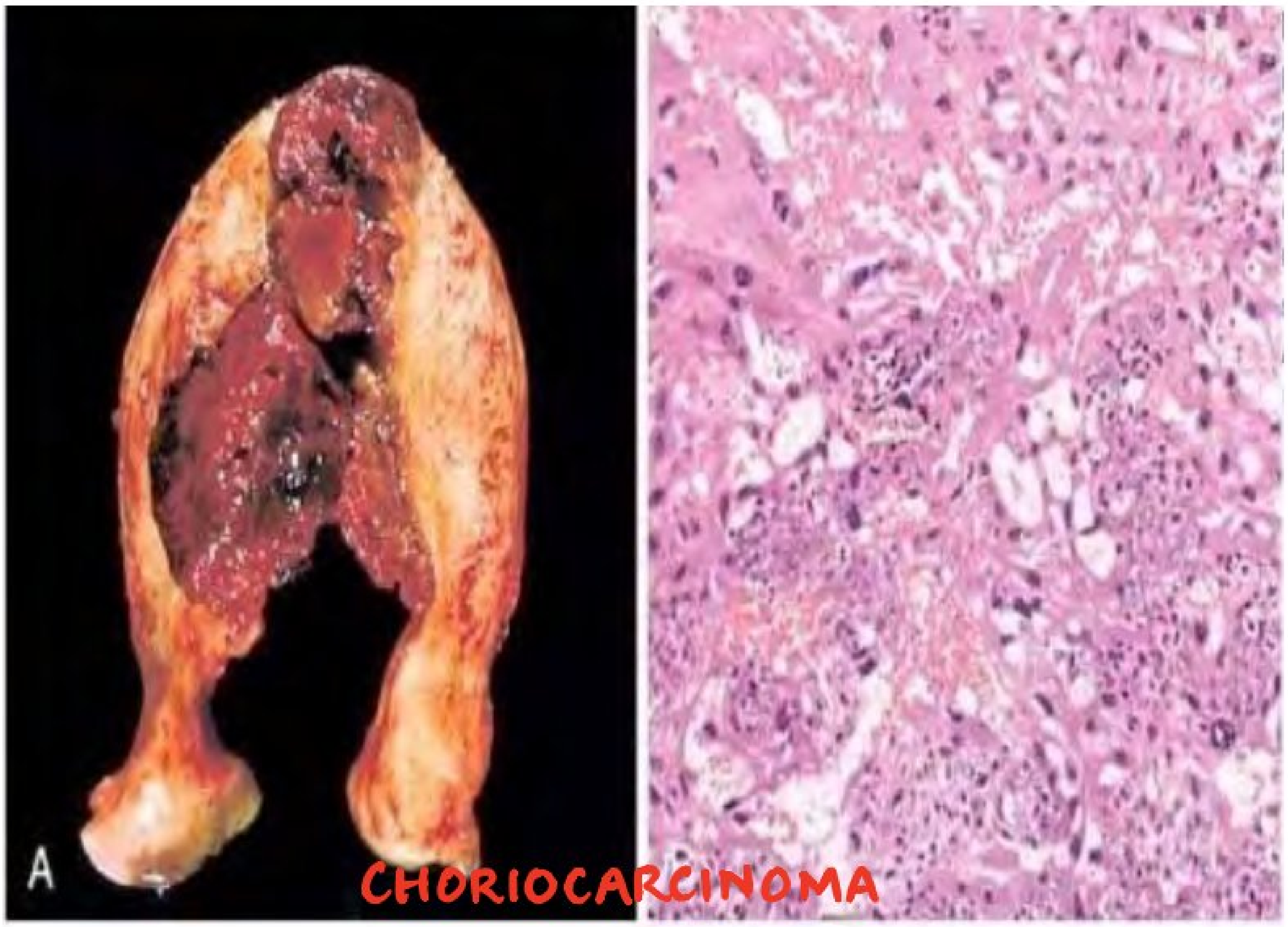

COMPLETE
PARTIAL
Complete hydatidiform mole
Partial hydatidiform mole
Coexistent mole and live fetus
INVASIVE
Invasive mole
CHORIOCARCINOMA
Choriocarcinoma
PSTT
Placental site trophoblastic tumor



SNOWSTORM



CANON BALL Mets



CHORIOCARCINOMA

On HPE [Gold Std. Investigation]

- INVASIVE MOLE - VILLUS ⊕
- CHORIOCARCINOMA - VILLI Absent

CHORIOCARCINOMA	PSTT
(MC) after **Non-Molar Pregnancy**	occur after
HCG ⊕	⊗ [HPL > PLAP]
Malignant	**70% - Benign** ; 30% - Malignant
Made up of **DIMORPHIC Cells** (Mononuclear and Multinucleated Cells)	**Only Mononuclear cells**
Hemorrhage and Necrosis - more common	
CHEMOSENSITIVE	**CHEMORESISTANT**
RxOC : **Chemotherapy**	RxOC : **HYSTERECTOMY**

GTN STAGING

- **Stage 1** : Confined to Uterus
- **Stage 2** : Outside uterus but c̄in pelvis (Vaginal mets)
- **Stage 3** : Metastasis to lungs → GOOD PROGNOSIS
- **Stage 4** : Metastasis everywhere

WHO SCORE [MODIFIED] for PROGNOSIS and TREATMENT

	GOOD PROGNOSIS [LOW SCORE]	POOR PROGNOSIS [HIGH SCORE]
1. Age	≤ 39 yrs	≥ 40yrs
2. HCG	$< 10^3$	$> 10^5$
3. Type of Antecedent Pregnancy	Molar	Full term
4. Duration from Antecedent Pregnancy	< 4months	≥ 12months
5. Tumor size	< 3cm	> 5cm
6. Site of Mets	Lungs, Vagina	Brain, Liver
7. No. of Mets	< 4	> 8
8. Any Prev. Chemo (Recurrence)	Single Agent	Multi Agent

TOTAL SCORE

1. If ≤6 → LOW RISK GTN → RxOC → c̄ METHOTREXATE (Single Agent Chemo) → if Resistant to Mtx → Give ACTINOMYCIN-D

2. If ≥7 → HIGH RISK GTN → MULTI AGENT CHEMO Rx Regimen → 1. EMACO Regimen - if Resistant give 2. EMAEP Regimen

E - Etoposide	- D_1	if Resistant →	E - Etoposide	- D_1
M - Mtx	- D_2		M - Mtx	- D_2
A - Actinomycin	- D_3		A - Actinomycin	- D_3
C - Cyclophosphamide	- D_4		E - Etoposide	- D_4
O - Oncovin	- D_5		P - Cisplatin	- D_5

Follow up by HCG ; weekly HCG - till we get 3 Ⓝ values
↓
Stop Chemo
↓
Then do Monthly HCG

- Total duration of Surveillance - 12 months

- Advice : Not to conceive , use OCP's

Antepartum Hemorrhage

Any bleeding which occurs in Genital tract or from genital tract beyond viability. (IN INDIA, Period of viability = 28 wks)

CAUSES

- Placental Causes (MC)
  - MC Abruptio Placenta
  - Placenta previa
  - Circumvallate placenta
  - Circummarginate placenta
- Fetal causes
  - Vasa Previa

PLACENTA PREVIA	ABRUPTIO PLACENTA
Placenta is located in lower uterine segment. For localization of Placenta; USG should done in 3rd Trimester Repeat scan: In minor degree → 36 wks In major degree → 32 wks	Placenta seperation of normally situated placenta
Incidence: 1 in 300 Pregnancy	Incidence: 1 in 200 Pregnancy
Recurrance Rate: 5%	Recurrance Rate: 12 to 15%
MC Risk Factor: Previous h/o Placenta previa 2nd MC: Previous h/o cesarean section	MC Risk Factor: Previous h/o Abruptio Placenta

Common Risk Factors for both Placenta previa & Abruptio placenta

1. Multiparity
2. ↑ Maternal Age
3. Smoking
4. Twin Pregnancy

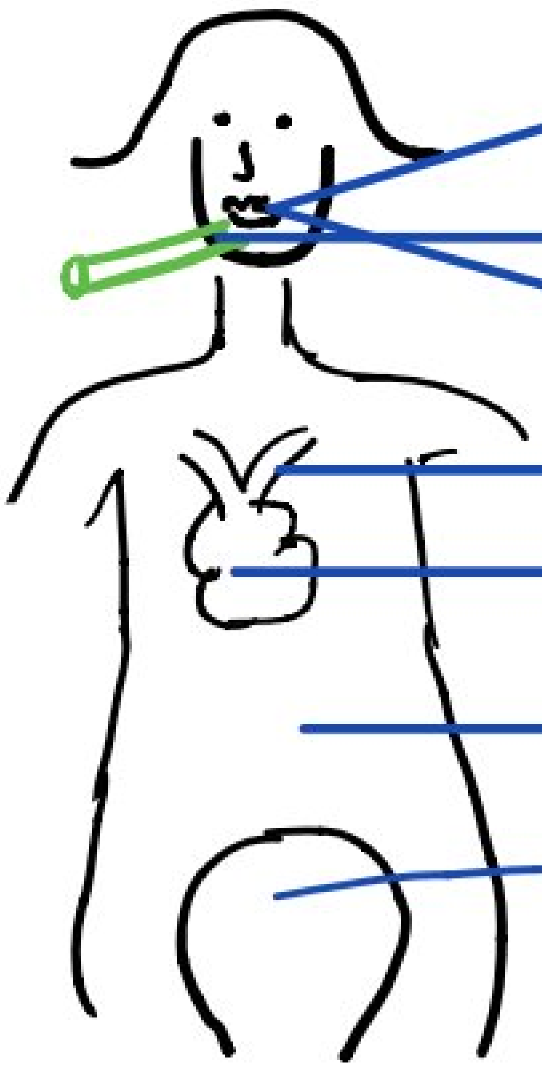
APH → MC in multiparous & smoking is a Risk Factor
PPH → MC in nulliparous & smoking is protective

Risk Factors

When Placenta is Big
1. Twin pregnancy
2. Diabetes
3. Succenturiate lobe
4. Placenta bilobata

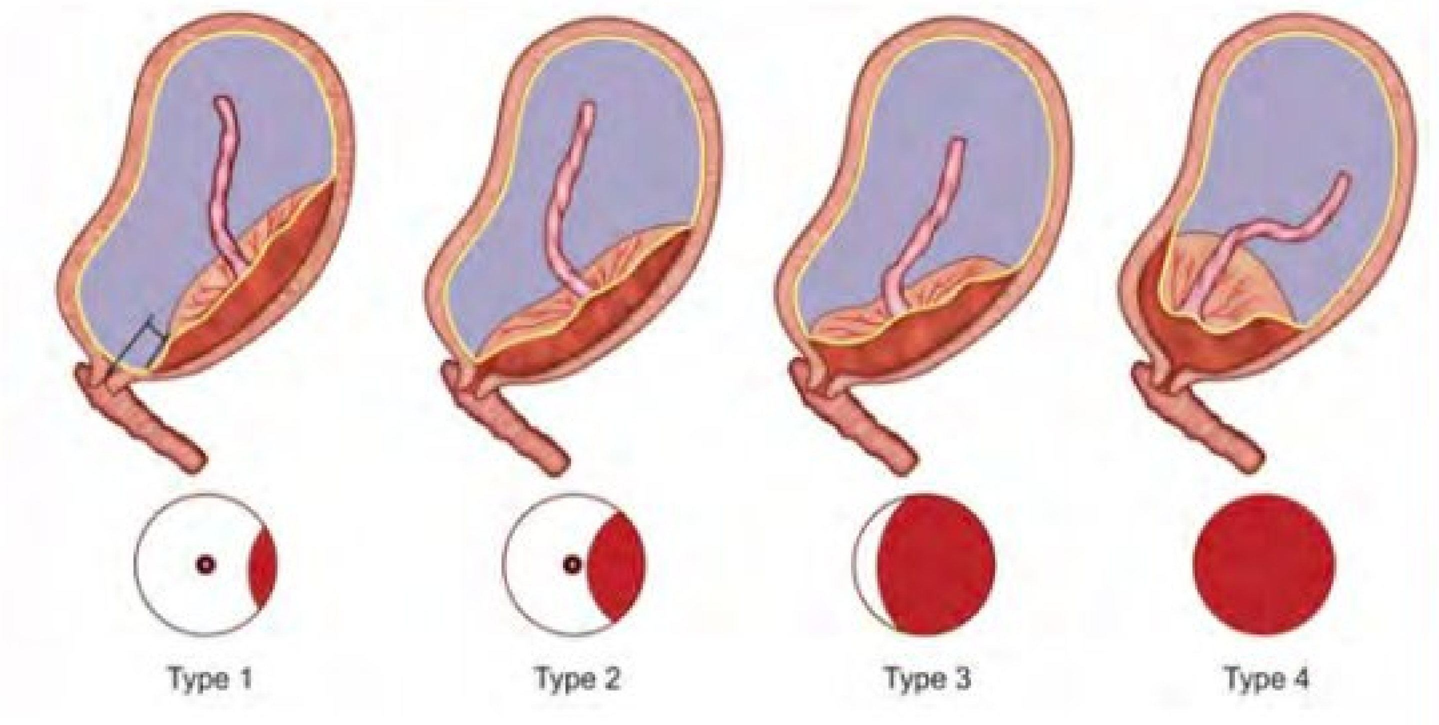
Endometrium is defective
1. Smoking
2. Endometriosis
3. Excessive curettage
4. Previous uterine Surgery
   → Cesarean Scar
   → Myomectomy Scar

Risk Factors

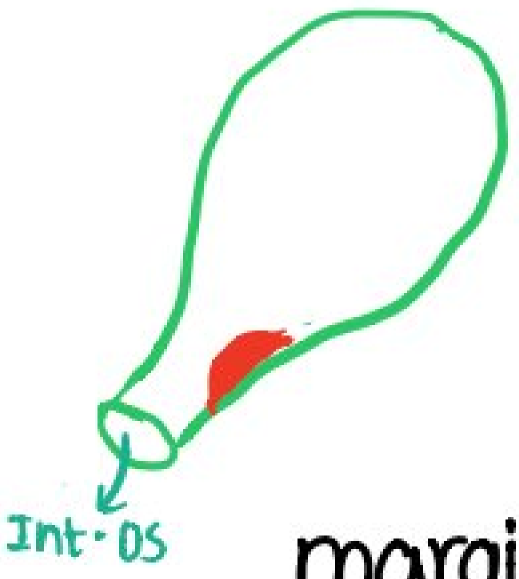


→ Smoking
→ Coccaine Abuse
→ FA deficiency
→ Thrombophilia
→ ↑ BP
→ h/o Abdominal Trauma
→ • Fibroid
• PROM
• Polyhydramnios
• Twin Pregnancy

CLASSIFICATION



TYPE 1



- Placenta in LUS
- Distance from internal os is <2cm
- Doesnot reach up to margin of internal os.

aka Lateral/Low lying placenta previa

CLASSIFICATION – Page Classification

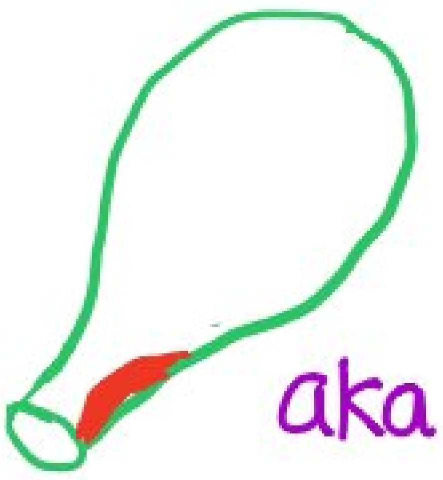
Grade 0 = Retrospective diagnosis based on Retroplacental Clot

Grade 1 = Pain in abdomen
(d/t thromboplastin, which is uterotonic agent)
+ Bleeding PV + FHS = Normal

Grade 2 = Pain in abdomen + Bleeding PV + Fetal distress/death

Grading 3 = Pain in abdomen + Bleeding PV + Fetal death + Patient is in shock + DIC
↓
d/t Release of thromboplastin

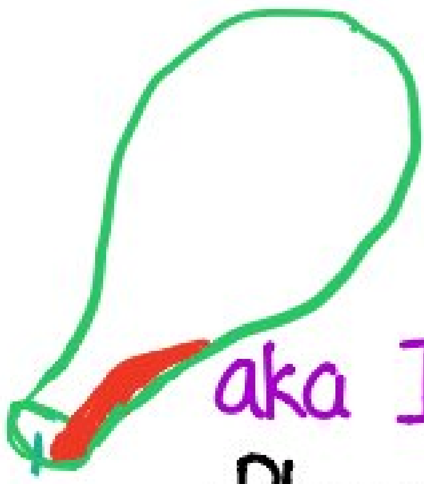
TYPE-2



-Placenta reaches upto margin of internal os

aka Marginal Placenta Previa

TYPE-3

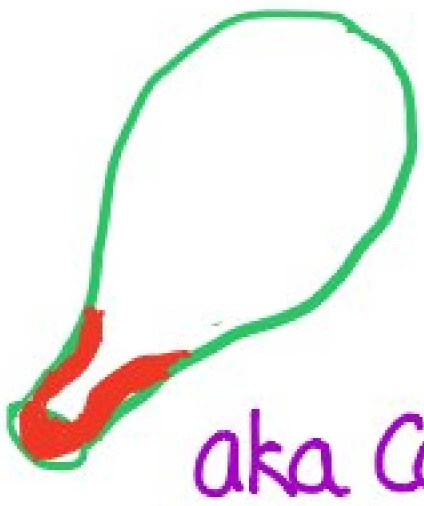


-Placenta partially covers internal os

aka Incomplete placenta previa

•Placenta doesnot cover dilated os.

TYPE-4



•Placenta completely covers internal os.

aka Complete /Central Placenta Previa

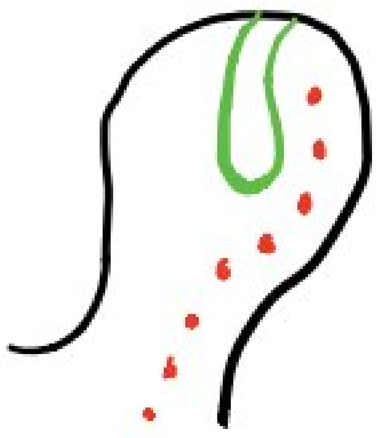
•Placenta covers dilated os.

In type 1 → Anterior / → Posterior

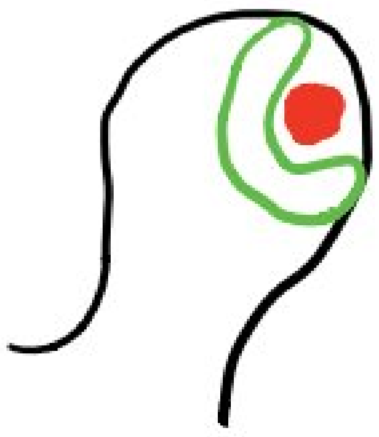
In type 2 → Anterior / → Posterior

Type 2 Posterior is called as Dangerous Placenta Previa, ∵ in type-2 posterior, Placenta located posteriorly, and it's reaching upto level of internal os. On USG, So many times it's missed as it lies posterior/ behind fetus, During Labor, when fetus goes down it presses against placenta & there will be bleeding.

Varities of Abruptio Placenta



REVEALED VARIETY



CONCELED VARIETY

↓

In this variety, Blood collected behind Placenta

↓

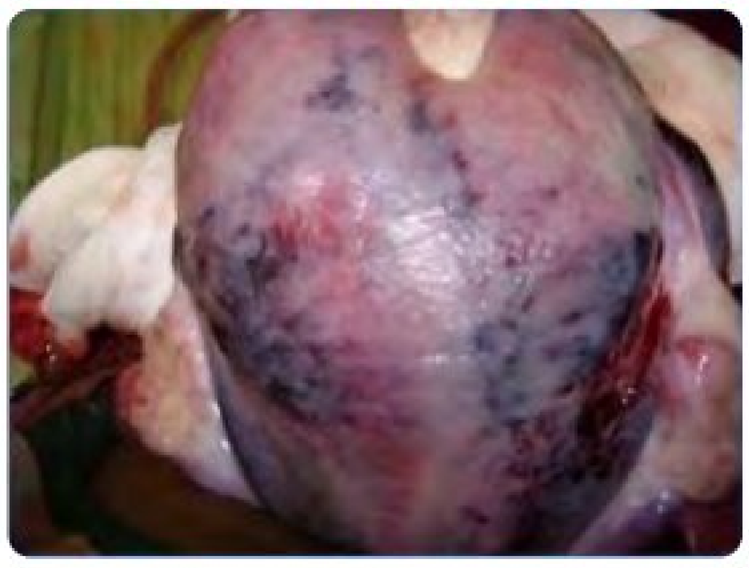
Enters myometrium of uterus

↓

Uterus Appears Bluish/ wine coloured

↓

COVELAIRE UTERUS



COVELAIRE UTERUS (Imp. Points)

1. Is not an indication for Hysterectomy
2. It's a Risk factor for PPH

MC variety of Abruptio Placenta is - MIXED VARIETY

∴ It's called as Dangerous Placenta Previa

Minor Degree Placenta Previa

↓

Type 1 → Anterior
→ Posterior

Type 2 only Anterior

Vaginal Delivery can be tried.

Major Degree Placenta Previa

↓

Type-2 : Posterior
Type-3
Type-4

Always do cesarean section

In all posterior varieties of Placenta Previa

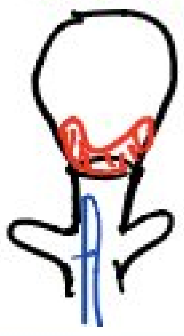
↓

Since there is less space (d/t sacrum)
Fetal distress on pushing Head into pelvis perabdominally

↓

STALLWORTHY SIGN

SYMPTOMS	SYMPTOMS
1. Bleeding in 3rd Trimester	1. Bleeding in 3rd Trimester
* Bright Red in colour	* Dark Red in colour
* Painless (No pain in Abdomen)	* Pain in abdomen is present
* Causeless	* Cause for Bleeding (Trauma ∴ aka Accidental Hemorrhage)
* Recurrent in same pregnancy	* Not recurrent in same preg.
* Warning Hemorrhage seen.	* No warning Hemorrhages

<table>
<tr><th>PLACENTA PREVIA</th><th>CONCEALED ABRUPTIO</th></tr>
<tr><td>General Examination:
General condition of patient is proportional to blood loss
P/A: * Uterus is soft, relaxed, non-tender FHS - easily heard
Fetal Parts: Easily felt
* Height of uterus = Period of gestation
* Fetal FHS present
P/V: Contraindicated in APH
Contraindicated in Placenta previa</td><td>General Examination:
General condition of patient is not proportional to blood loss.
P/A: * Uterus is hard, tender, tensed
FHS - Not easily Heard
Fetal parts : Not easily felt
* Height of uterus > Period of gestation
* Fetal distress/death</td></tr>
<tr><td colspan="2">IOC (for APH):- TAS ⇒ Screening test to differentiate b/w Placenta previa & Abruptio Placenta</td></tr>
<tr><td>IOC in Placenta Previa is ⇒ TVS

Probe doesnot cross internal os</td><td>IOC in Abruptio Placenta ⇒
Clinical > TAS</td></tr>
<tr><td>Malpresentations are more common in placenta previa
↓
Transverse lie > Breech</td><td></td></tr>
</table>

MANAGEMENT OF ABRUPTIO PLACENTA

Principles :

i. Never wait & watch

Interval b/w abruptio & delivery plays an very important role in development of DIC. More the interval, more the chances of DIC.

ii. Never give Tocolytics

iii. Always best management → TERMINATION OF PREGNANCY

Mode of Labor

Fetus Alive

- If vaginal delivery is about to happen
  - ↓ Vaginal Delivery
- Otherwise cesarean section

Dead Fetus

Prefer vaginal delivery. But in some cases, Cesarean may be needed

VAGINAL DELIVERY IN ABRUPTIO

MISOPROST is C/I for induction of Labor

↓

In Abruptio, as it's tone of uterus ↑, with MISOPROST use

↓

↑ chances of Tachysystoles

↓

Leads to fetal distress

Tachysystoles → >5 contractions in 10 min

OXYTOCIN can be used to augment Labor

↓

No data which indicates that oxytocin can augment release of Thromboplastin into maternal circulation

ARM Hastens Delivery

No evidence to support this

If a patient of Abruptio develops DIC

↓

First correct DIC ⇒ f/b vaginal delivery

MANAGEMENT OF PLACENTA PREVIA

Terminate the pregnancy immediately

↓

ACTIVE MANAGEMENT

↓

In any condition, where mother life is at Risk

Continue the Pregnancy

↓

EXPECTANT MANAGEMENT

↓

Purpose : To let the fetus attain Lung maturity (37 wks)

Condition : Never putting mother Life at Risk

ACTIVE MANAGEMENT	EXPECTANT MANAGEMENT
• Hemodynamically unstable pt.	• If pt is hemodynamically stable.
• If continous bleeding present	• If the bleeding has stopped
• If gestational age ≥37 wks / if patient in Labor	• Gestational age is ≤36 wks
• If fetal distress is present	
• If on USG : Congenital anomaly in the fetus which is incompactible with life (Anencephaly)	• If on USG : Normal fetus & congenital anomaly compactible with life (Polydactyly)

Expectant management of Placenta previa is known as ⇒

Mcafee & Jhonson regime :

i. Admit the patient
ii. Arrange for blood
iii. If Rh -ve female → give Anti-D (300 mcg)
iv. Inj. CORTICOSTEROID (to attain lung maturity)
v. Tocolytics (if contractions ⊕)
(BEST : NIFEDIPINE)

Cervical Cerlage : Not a component of expectant management of Placenta Previa

Expectant Management

↓ Carry till 37 wks

↓ if fetal distress ⊕
Bleeding episodes ⊕
↓
Terminate immediately

Mode of Delivery

↓ Minor Degrees
↓
Vaginal Delivery

↓ * Major degrees of placenta previa
(Type 2 Posterior, Type 3, Type 4)
* Fatal distress
* Severe bleeding
(irrespective of type of Placenta previa)
↓
Cesarean Section

For mode of delivery in Placenta previa, it is necessary to know type of Placenta previa

↓ USG

↓ Double set up Examination

In OT, after doing all arrangement for cesareon section
↓
Do a P/V Examination
↓
k/a Double setup examination

DIC

Consumptive coagulopathy (all Clothing factors are consumed)

Obstretical causes of DIC

- Abruptio placenta (MC)
- IUD of fetus
- Amniotic fluid embolism
- Septic abortion
- Severe pre eclampsia/ eclampsia/HELLP Syndrome

Diagnosis

- S. fibrinogen (Clotting F. 1) = ↓
- Fibrin degradation product = ↑
- Di-dimer

Drugs in management of DIC

- Blood transfusion
- FFP
- Cryoprecipitate
- Platelet transfusion
- Heparin +/-

Drugs which are no role in DIC are,

i. EACA
ii. Tranexamic acid

Recombinant Factor VIII A : Role in DIC is uncertain

Q Which of the following is not used in DIC?

AIIMS 90

✓a) Heparin
b) EACA
c) Blood transfusion
d) Intravenous

Q A primipara whose period of amenorrhea is 30 wks develops sudden onset of sharp continous abdominal pain, shock & tender uterus without vaginal bleeding. She is not in labour.

What is the most likely diagnosis?

a) Amniotic Fluid Embolism
[✓ b) Placental Abruption]
c) Red degeneration of fibroid
d) Rupture of the uterus

Q At 28 wks USG (TVS) a G2 P1 female was detected as having major placenta previa. A confirmatory should be preformed

[✓ a) At 32 wks]
b) At 34 wks
c) At 36 wks
d) At onset of Labour

Q All are causes of APH except,

AIMPG 04

a) Placental previa
b) Abruptio placenta
c) Circumvallate placenta
[✓ d) Battle dore placenta]

Q Placenta previa Risk factor is a/w all of the following except.

AIPG 98

a) Large Placenta
b) Previous cesarean scar
[✓ c) Primi gravida]
d) Previous placenta previa

Q Placenta previa is characterised by all except:

a) Painless Bleeding
b) Causeless Bleeding
c) Recurrent Bleeding
[✓ d) Present after 1st trimester]

Q Regimen followed in expectant management of placenta previa

a) Liley's method
b) Crede's method
[✓ c) McAffee & Jhonson regime]
d) Brandt - andrews method

Q Expectant management of Placenta previa includes all except

a) Anti - D
[✓ b) Cervical Encirclage]
c) Blood transfusion
d) Steroids

Q A primagravida at 37 wks of gestation reported to labor room c̄ central placenta previa c̄ high bleeding per vaginum. The fetal HR was normal at the time of examination. The best management option for her is

AIPG 03

a) Expectant management
[✓ b) Cesarean section]
c) Induction & Vaginal delivery
d) Induction & Forceps delivery

POST PARTUM HEMORRHAGE

3rd Stage → Placental Separation Avg. time 15-20 min, if >30 min Prolonged

- Uterine contractions are required for placental separation from decidua basalis

Signs of Placental Separation

1. Gush of Blood → opens up the venous sinuses present in myometrium

   Middle layer muscle fibres are arranged in "CRISS CROSS FASHION" - "LIVING LIGATURE"

   So, when middle layer contracts - constricts vessel
2. Suprapubic Bulge
3. Lengthening of cord

   ↓

   lengthening of cord - its only apparent and permanent lengthening
4. Fundal Height will increase - placenta pushes uterus up

BEST SIGN - Placenta lying in VAGINA f/b Lengthening of cord

PLACENTA

Ⓝ Placental Wt = 500g

Ⓝ Volume = 500ml

Ⓝ Diameter = 20cm

Avg. thickness = 2.5cm

Placentomegaly : if >4cm THICK

↳ causes : 1. Hydrops fetalis

2. Intrauterine Infection (Syphilis / CMV)

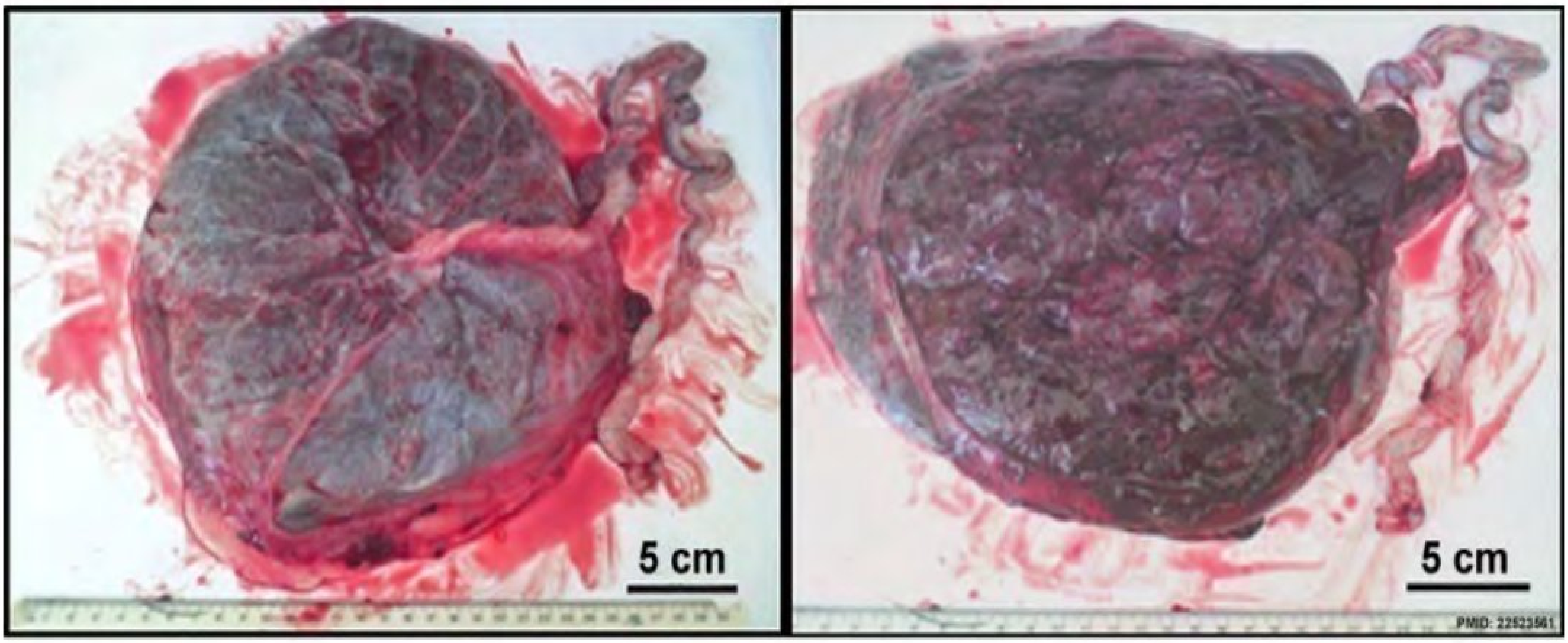
3. DM

4. Multifetal Pregnancy

MATERNAL side : side which faces DECIDUA BASALIS
↓
Bed of Placenta

Lobes : 15 - 20 each, each contain multiple lobules

Term Placenta



Fetal side

Maternal side

Lobules : are the functional unit of placenta

Tissue of Placenta formed by **CHORION FRONDOSUM**

Placenta = Decidua Basalis + Chorion frondosum → lined by : TROPHOBLAST CELLS

FETAL SIDE :

- when umbilical cord is attached @ centre
- Smooth and shiny covered c̄ membranes ⟶ innermost memb: AMNION

AMNION : 1. Avascular memb

2. Derivative of Fetal ectoderm

Outer memb. : CHORION

Primitive Hb

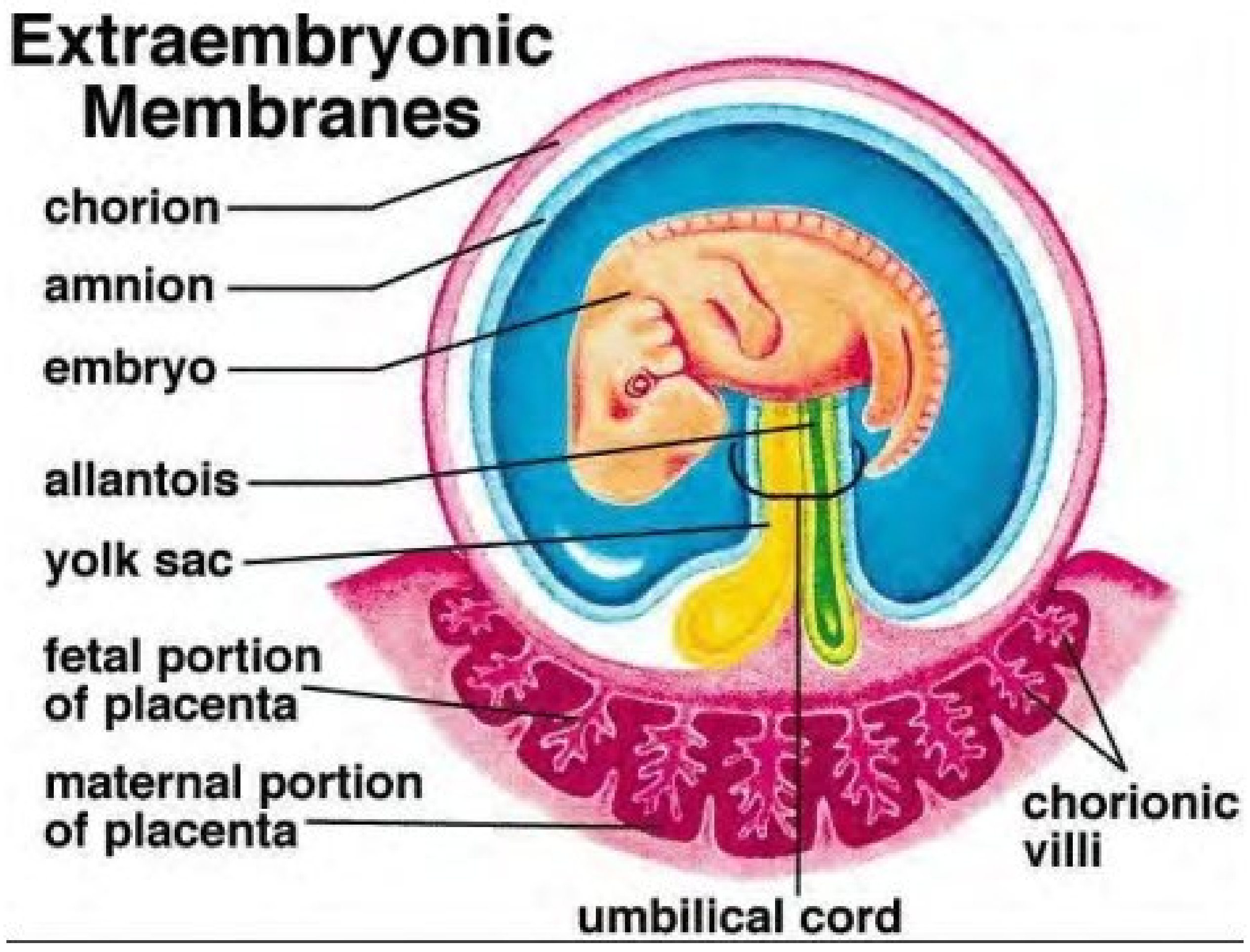
GOWER-1

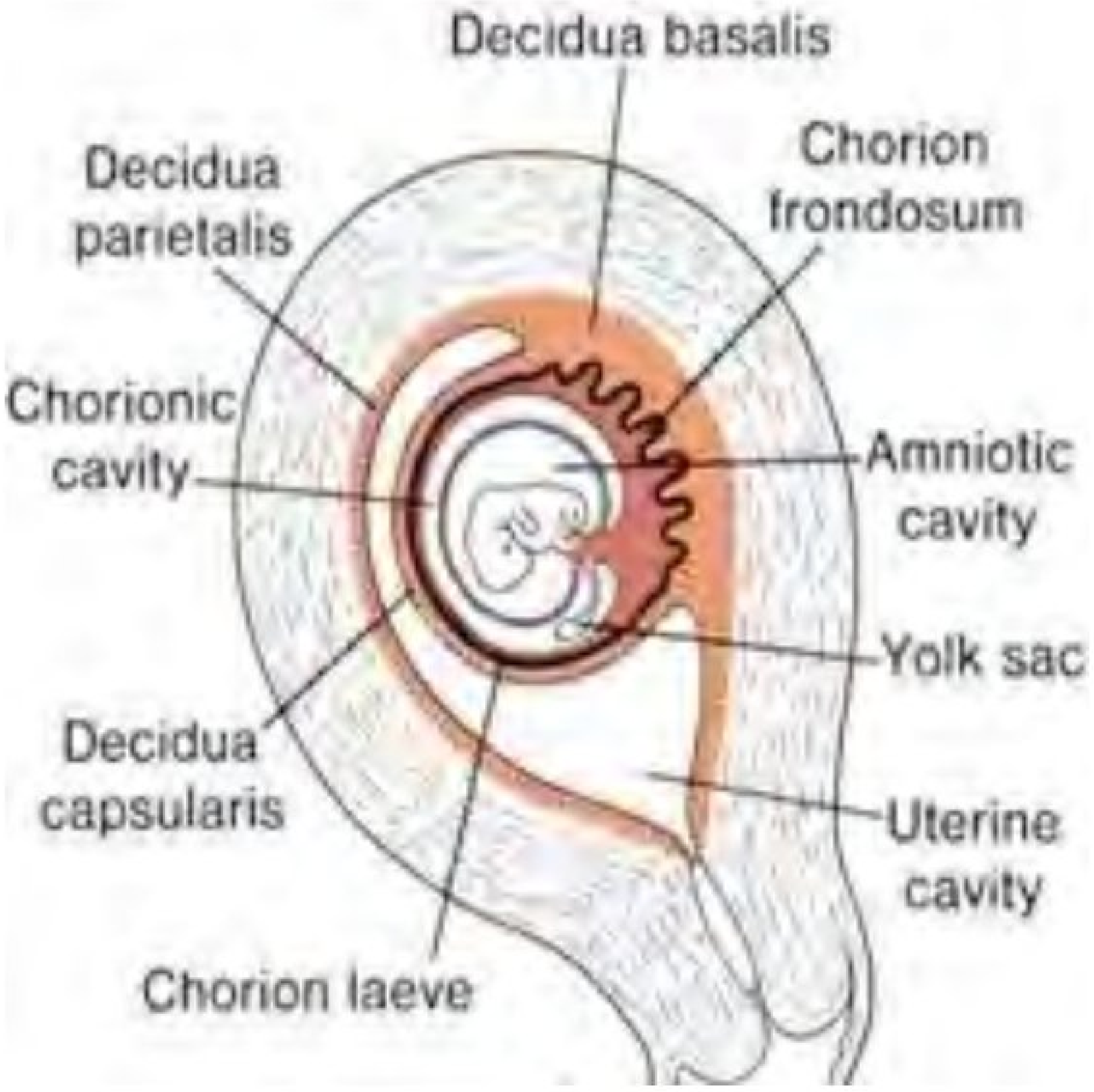
GOWER-2

PORTLAND

These are **4 Fetal membranes** :

1. **AMNION**
2. **CHORION** - forms placenta
3. **YOLK SAC** - 1st site for Fetal Hematopoiesis begins 3-6 wks
   - after 6wks - mainly @ LIVER → HbF formed
   - beyond 20wks - occurs only in long bones
4. **ALLANTOIS** - its a diverticulum from HINDGUT that grows into connecting stalk
   forms umbilical vessels





1. **PLACENTA SUCCENTURIATA** - Accessory lobe (small) attached to main lobe
2. **PLACENTA BILOBATA** - Cord attached to both lobe
3. **PLACENTA BATTLEDORE** - cord attached to periphery
4. **VELAMENTOUS PLACENTA** → further type

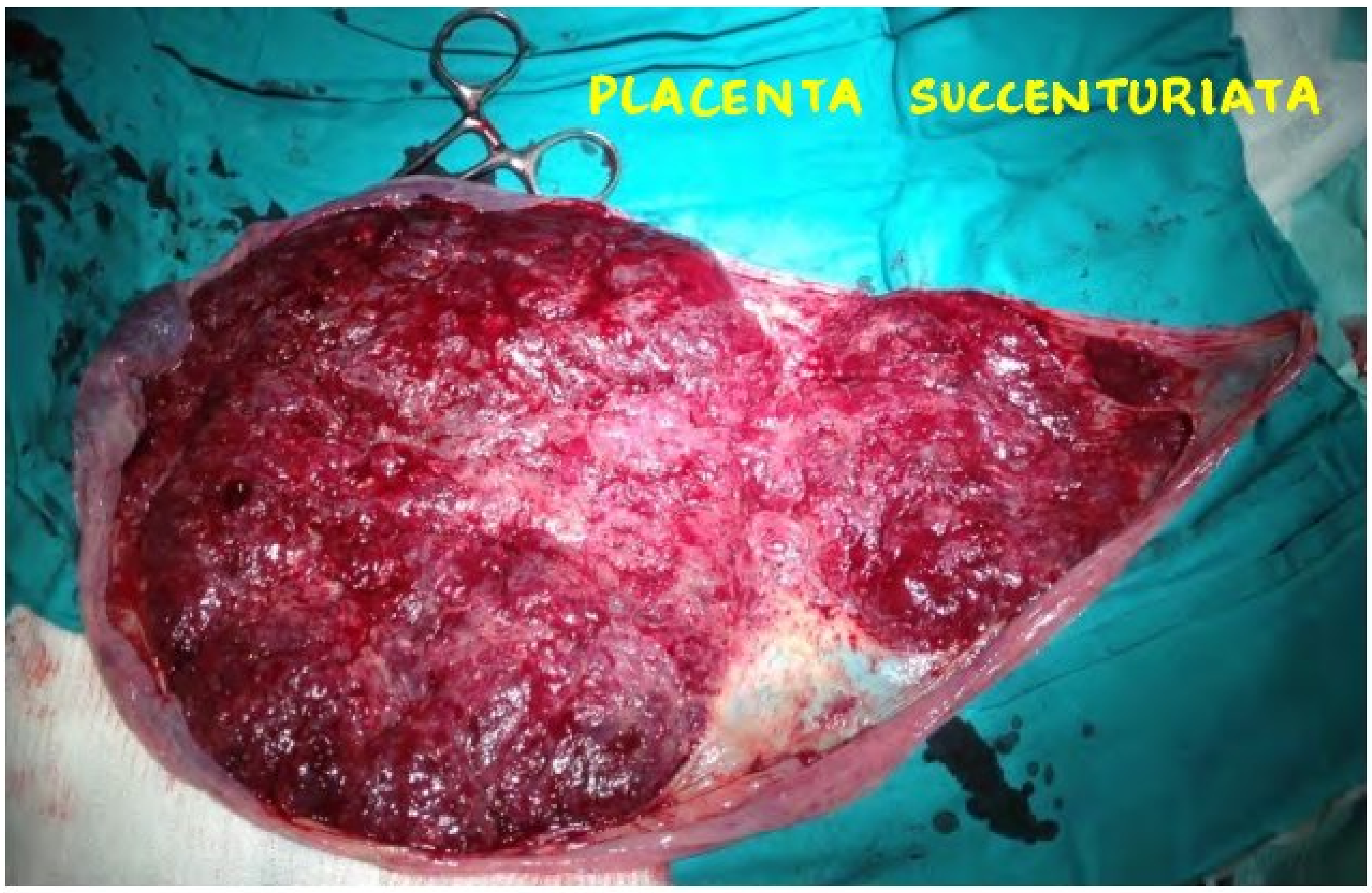
VASA PREVIA - blood vessels are travelling over internal os

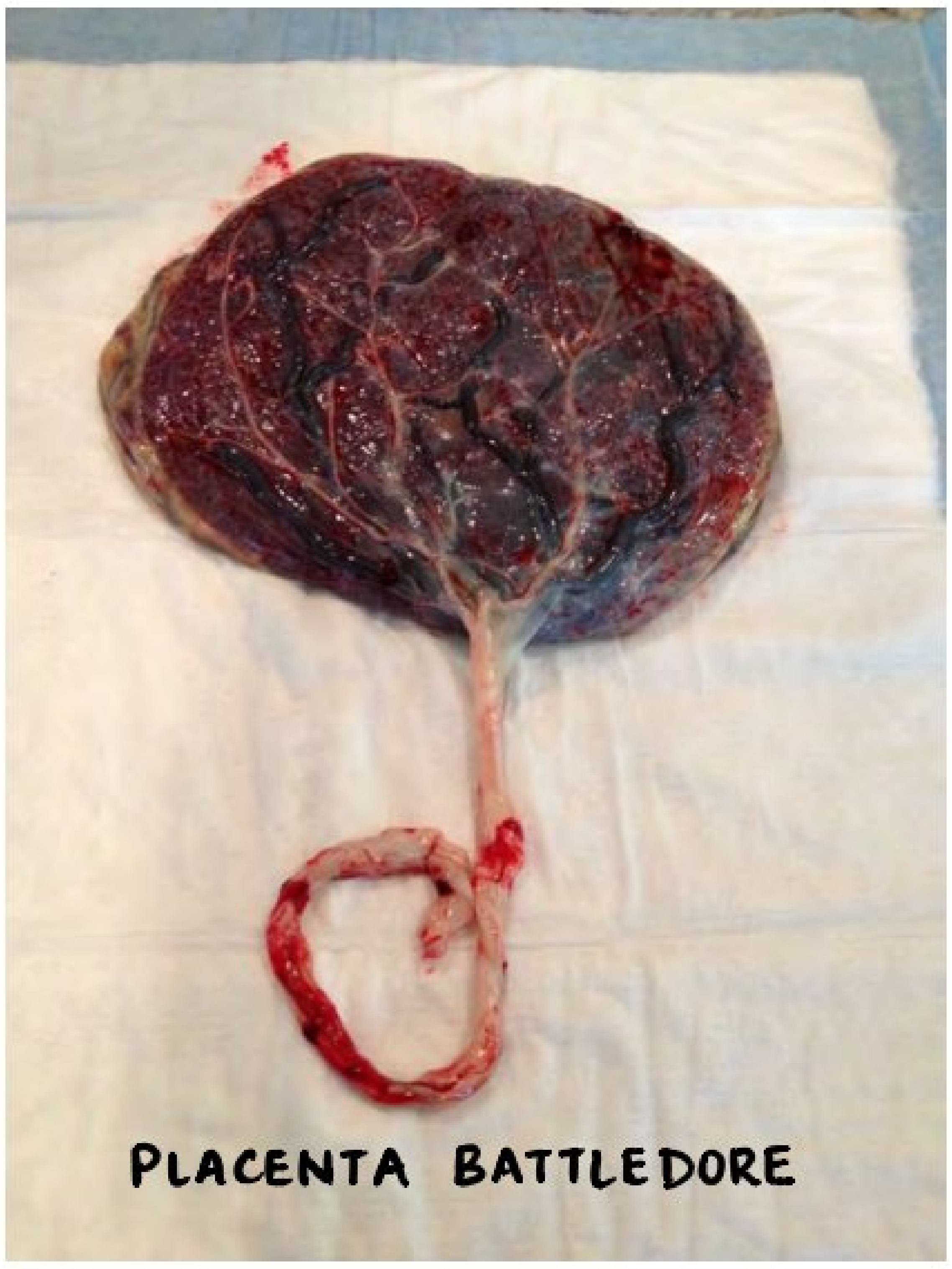
So when memb. rupture - fetal blood vessels also rupture

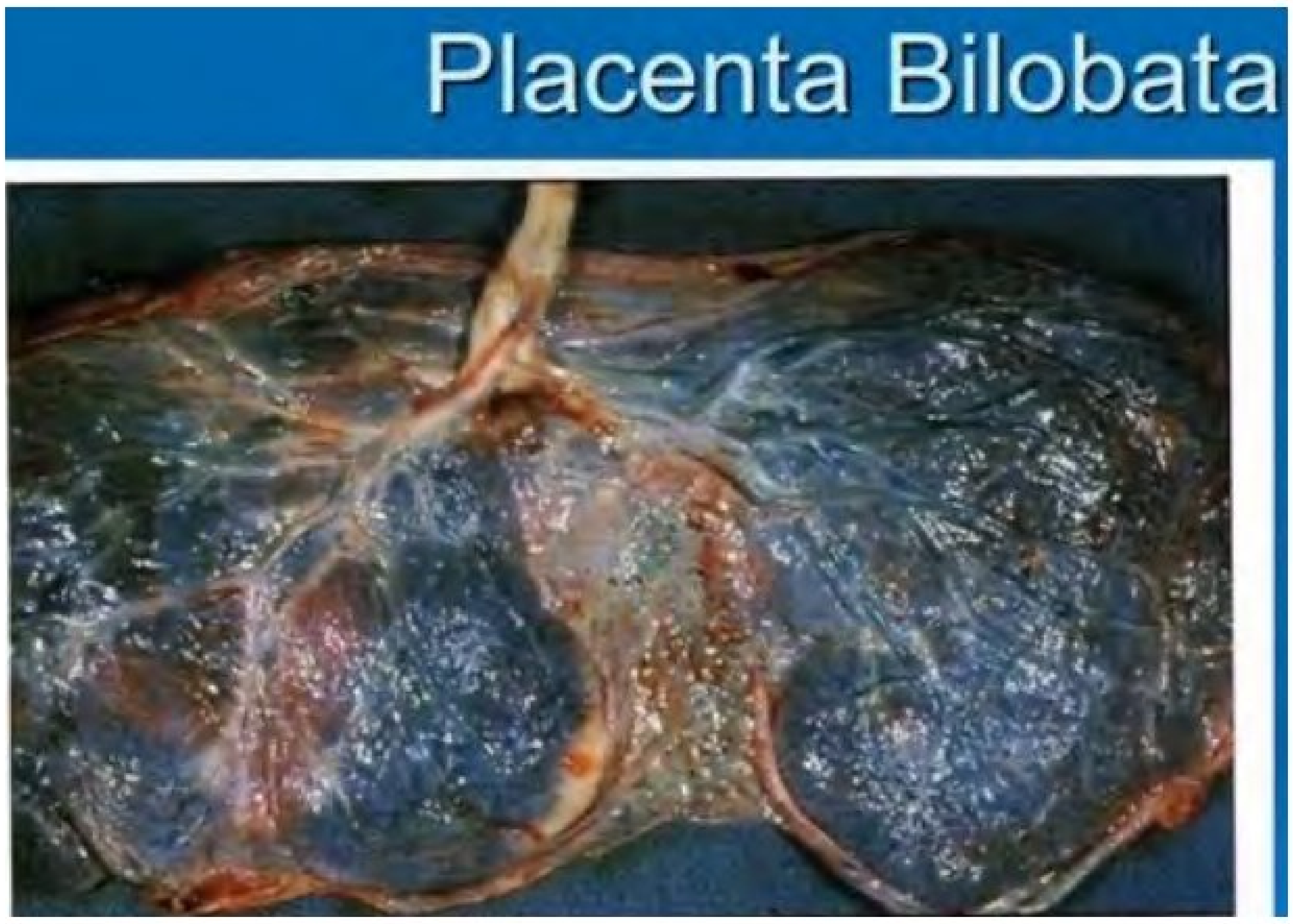
↓

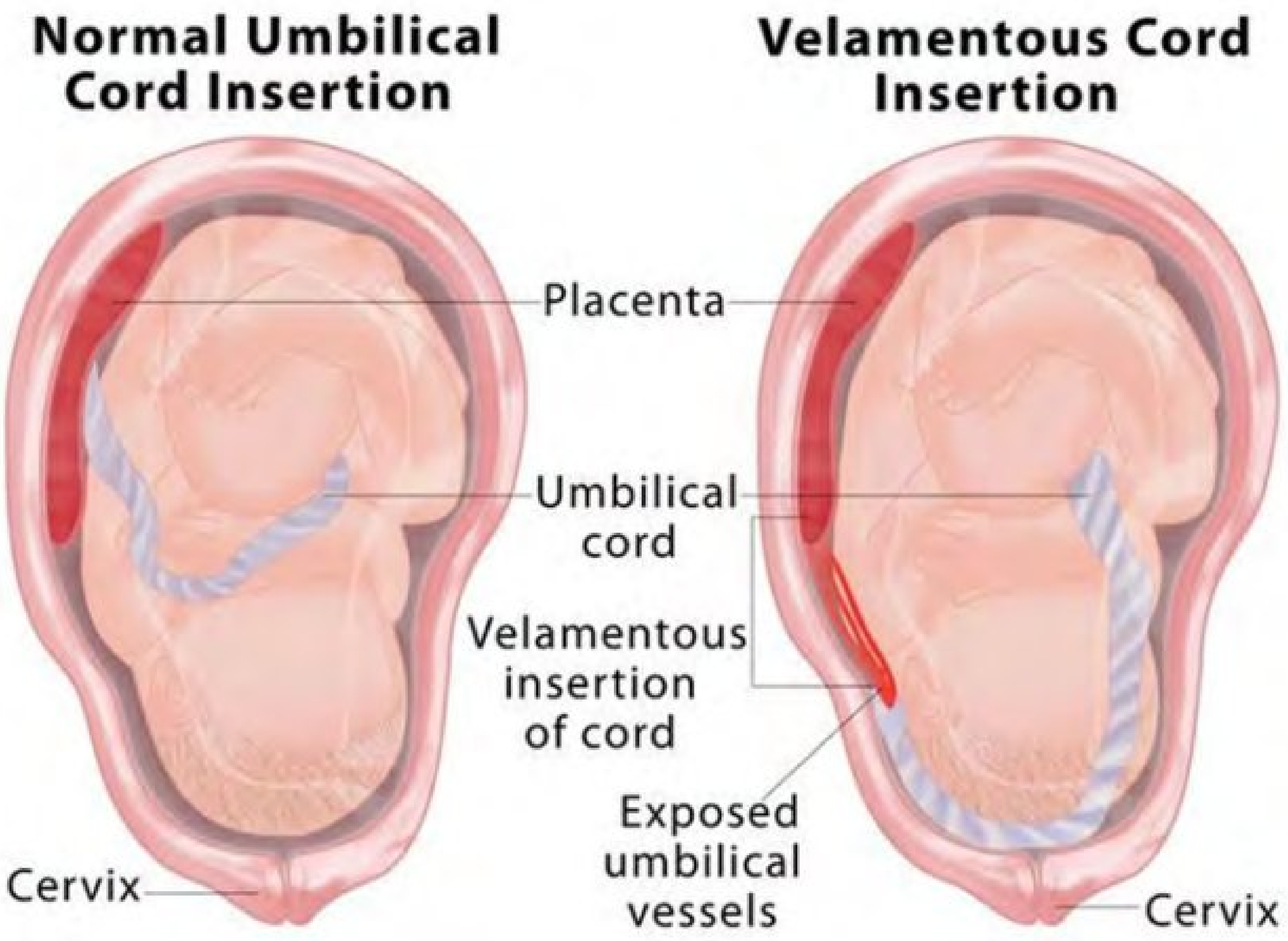
cause Bleeding

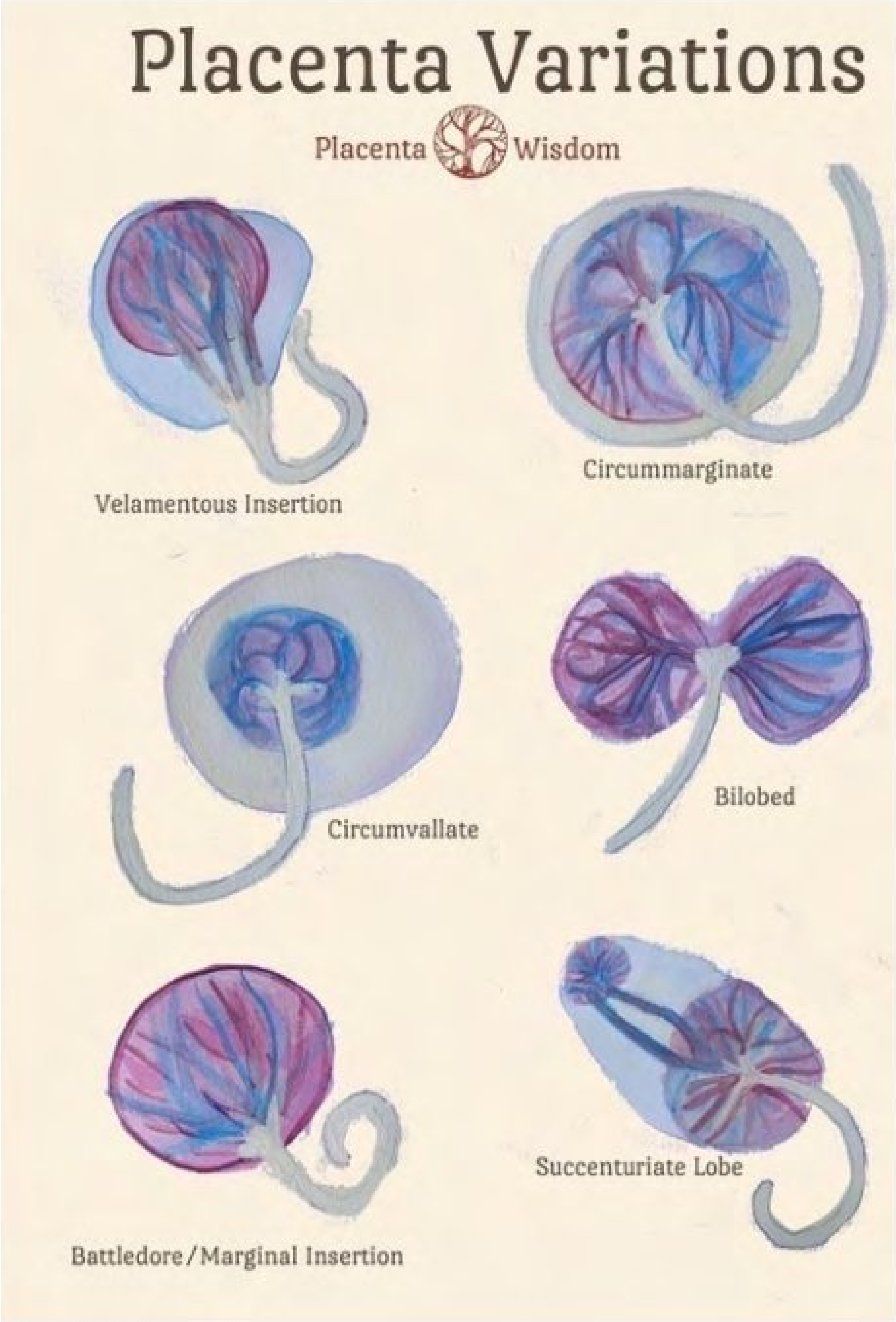
This bleeding is [APH] → Fetal Blood loss → a/w severe fetal distress
↓
OBSTETRIC EMERGENCY and EMERGENCY C·S done


PLACENTA SUCCENTURIATA


PLACENTA BATTLEDORE


Placenta Bilobata

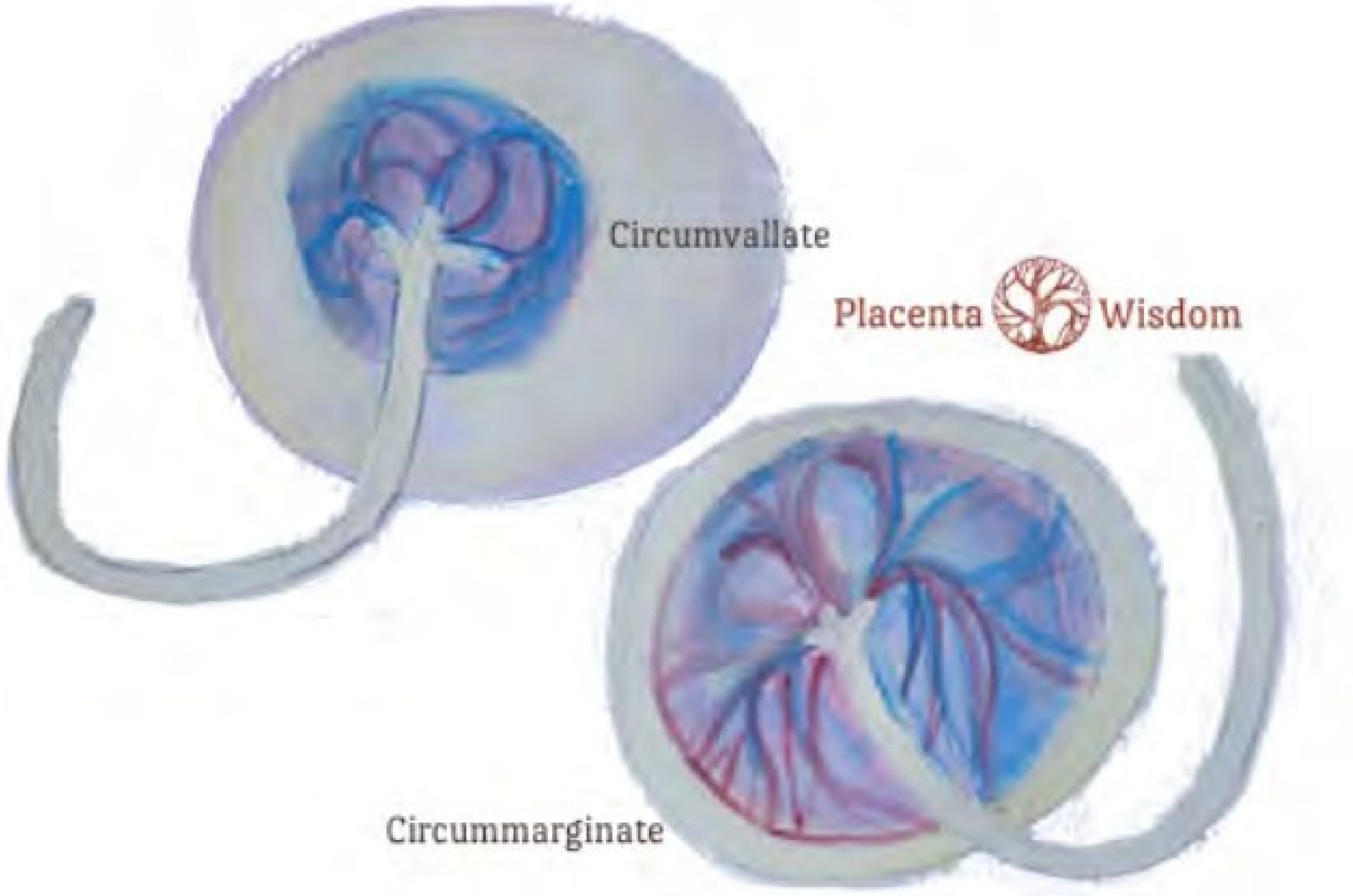

Normal Umbilical Cord Insertion
Velamentous Cord Insertion
Placenta
Umbilical cord
Velamentous insertion of cord
Exposed umbilical vessels
Cervix
Cervix



How to differentiate blood is Fetal or Maternal ?

1. **KB Test** (KLEIHAUER BETKE Test)

- to differentiate fetal RBC from maternal RBC
- LAB Test
- ACID Denaturation test (Fetal blood is resistant to acid denaturation)
- CITRIC ACID is used.
- smear is made of maternal blood and is exposed to acid
- QUANTITATIVE TEST
- done in Rh NEGATIVE Pregnancy



count RBC → formula → Amount of Anti-D dose calculated

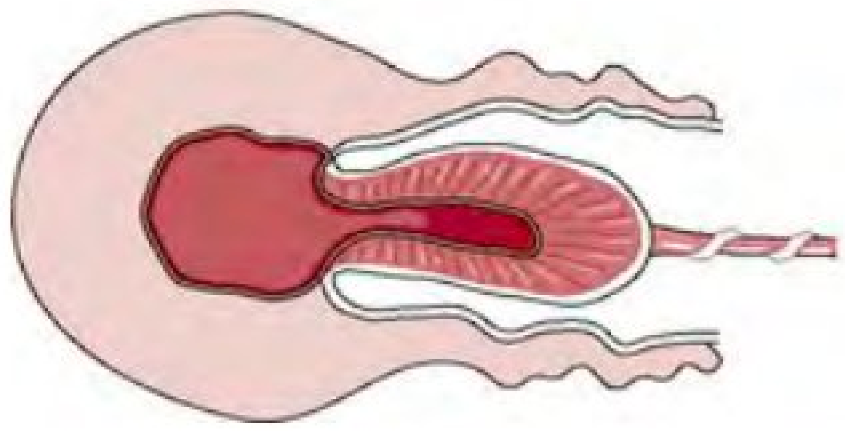
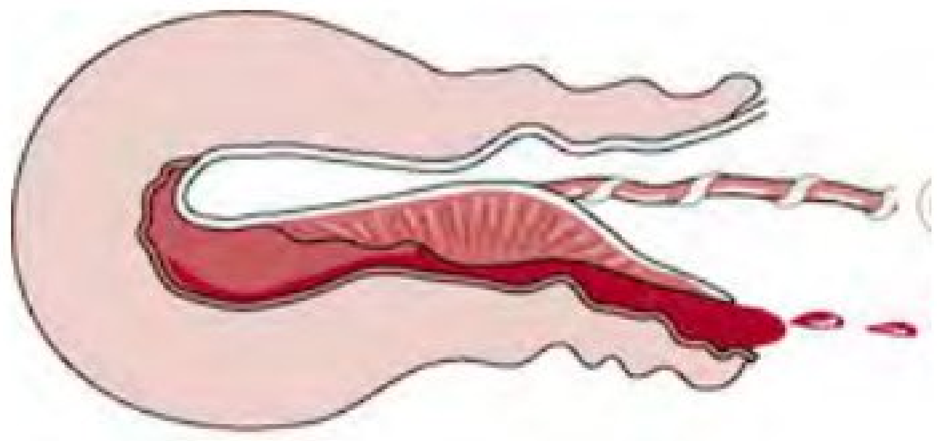
CHORIONIC PLATE SMALLER THAN BASAL PLATE
- if margin elevated → CIRCUMVALLETE PLACENTA
- if margin is not elevated b/w chorionic plate and Basal plate → CIRCUMMARGINATE PLACENTA

2. **APT Test done** (Alkali Denaturation test)

- 1% NaOH is used : Fetal Blood is Resistant to alkali
- Bedside test
- Therefore only a QUALITATIVE Test

PLACENTAL SEPARATION

SCHULTZ METHOD	DUNCUNS METHOD
• Separation starting from centre	• separation starting from periphery
• MC	• LC
• Collection of blood behind the placenta RETROPLACENTAL CLOT (RPC)	• External bleeding will begin ≤ separation

• External blood loss is seen after complete separation • TOTAL BLOOD LOSS – LESS • Fetal side presents @ vulva – "SHINY SCHULTZ" 	• MORE • Maternal side presents @ vulva "DIRTY DUNCANS" 

Ⓝ Blood loss in Vaginal delivery = 500ml

C.S = 1000ml

In PPH – Blood loss – Vaginal delivery
- >500ml – MILD
- >1000ml – SEVERE

CS – >1000ml

ACOG → PPH >1000ml irrespective of mode of delivery

PPH – any blood loss which produces SIGNS and SYMPTOMS of HYPOVOLEMIA

PRIMARY PPH	SECONDARY PPH
• if blood loss happens ≤in 24 hrs of delivery • (MC) cause : Uterine atony PLACENTA SUCCENTURIATA	• if blood loss happens beyond 24hrs of delivery (MC) cause : Retained Placental tissue

(MC) cause of PPH – 4T's

Atony > Trauma to Genital tract > Retained placental tissue > Thrombosis (defective coagulatin)

RISK FACTORS for PPH

1. Polyhydraminos
2. Multifetal gestation
3. Macrosomia
4. Prolonged labour
5. Augmentation of Labour
6. Instrumental delivery
7. Multiparous > Primi
8. Intra-uterine infections - CHORIOAMNIONITIS
9. Fibroids
10. DIABETES MELLITUS
11. PRE-ECLAMPSIA
12. ANTE - PARTUM HEMORRHAGE

PREVENTION PROPHYLAXIS FOR PPH

AMTSL (Active Management of 3rd Stage of Labour)

WHO - 4 Components

1st Component : Give uterotonic agent immediately after delivery

↓

done ≤in 1min of delivery

Uterotonic Agents

DOC : OXYTOCIN 10IU → 1) i.m bolus or
2) i.v infusion

NEVER AS I.V BOLUS - if given can cause Severe Hypotension →
1. Tachycardia
2. Arrhythmia
3. M.I
4. Cardiac arrest

iv infusion has immediate onset

im bolus delayed onset by 3min

Natural OXYTOCIN Synthesized from PARAVENTRICULAR NUCLEUS of HYPOTHALAMUS

- its a NONAPEPTIDE
- Stored in POSTERIOR PITUITARY
- Short t½ : 3min (3-5min)

SYNTHETIC OXYTOCIN

- its an OCTAPEPTIDE
- cause : PHYSIOLOGICAL UTERINE CONTRACTION (every contraction f/b Relaxation)

OXYTOCIN

- act equally on UPPER SEGMENT as well as LOWER SEGMENT of the uterus
- cause Ca^{2+} influx ⟶ Main MOA
- also causes Release of $PGF_{2\alpha}$ from decidua and cause contraction of smooth muscles

2nd Drug : METHERGINE (Methyl Ergometrine) 0.2mg i.m injection

S/E : SEVERE TRANSIENT HYPOTENSION

C/I : 1. Pre-Eclampsia
2. Eclampsia
3. Heart ds
4. Peripheral Vascular Ds
5. Rh Negative Pregnancy
6. Delivery of 1st TWIN
- acts more on LUS so given after delivery of 2nd TWIN

3rd Drug : SYNTOMETRINE (5 IU Oxytocin + 0.5mg Methergine)

4th Drug : CARBTOCIN - Synthetic Oxytocin Analogue

- its an OCTAPEPTIDE
- t½ = 40min
- 100mg slow i.v over 1 min

5th Drug : MISOPROSTOL [PGE_1] → Tablets give Miso when parenteral drugs can't be given

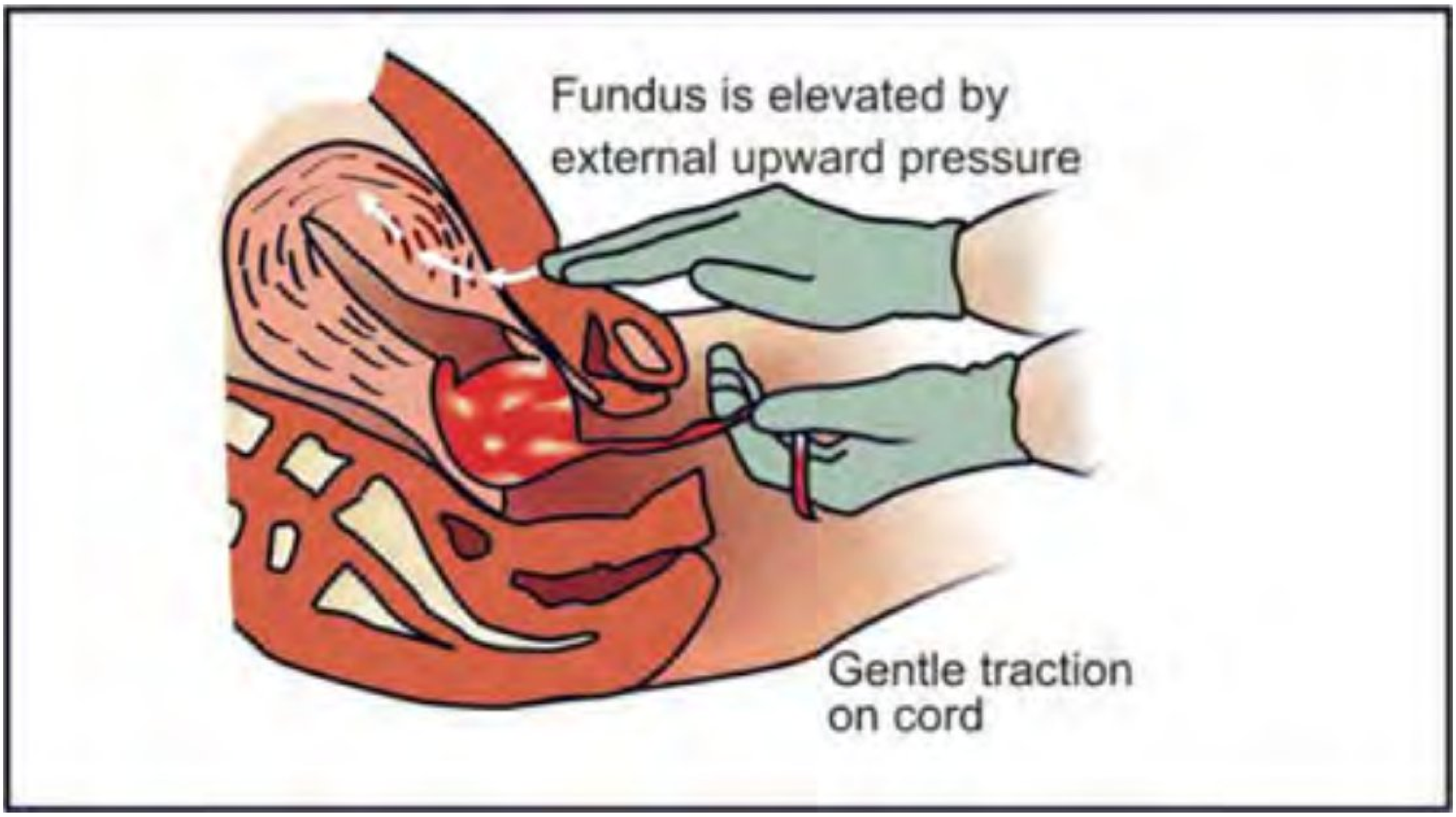
in HIGH RISK Pts ⟶ OXYTOCIN + MISOPROSTOL
also add TRANEXAMIC ACID (Anti-fibrinolytic)

For Prophylaxis of PPH : 600mg per oral [WHO]
↳ P/R not preferred route
↳ But Advantage : MINIMISE S/E

S/E
1. HYPOTENSION
2. Nausea, Vomiting
3. Fever ≤ chills — most important

2nd Component : Delivery of Placenta by controlled cord traction

" Modified Brandt Andrews Method" - to prevent Uterine invasion



3rd Component : Delayed Cord Clamping i.e >60sec (1min of delivery)

80ml of blood goes to baby in Delayed Cord Clamping

if done ≤ 1min → Early Cord Clamping
↓
Indications

1. If Baby needs Resuscitation
2. Rh Negative Pregnancy
3. HIV ⊕ Pregnancy
4. Baby k/c/o of Heart ds

4th Component : INTERMITTENT UTERINE TONE ASSESSMENT

(Uterine Massage is not a Routine Component of AMTSL)

MANAGEMENT OF PPH

NON-SPECIFIC

1. Secure 2 large bore i.v canula
2. i.v fluids (CRYSTALLOIDS)
3. Catheterise Bladder
4. Arrange Blood and Blood pdts
5. Investigations to be done

   HCt / Blood grp Rh / Coagulation Profile

SPECIFIC : done to stop bleeding

1. Start BIMANUAL UTERINE MASSAGE + Help
2. Start Uterotonics

   DOC : OXYTOCIN - 20 IU in 500ml NS/RL

   (Not in 5% DEXTROSE - because ADH like action of oxytocin given in Electrolyte deficient media - long duration)

   S/E : WATER INTOXICATION

   ↓

   if still Bleeding, then give

   METHERGIN / CARBOPROST - 0.2mg i.m

CARBOPROST [aka DINOPROST, HEMABATE]

- Methyl $PGF_{2\alpha}$ - 0.25mg given as i.m
- C/I in ASTHMA
- Most potent drug ; Repeat every 15-30min upto 8 doses in 24hrs (So max^m dose - 2mg)

MISOPROST

- is an add on drug which can be given c̄ OXYTOCIN
- For Rx of PPH : 800mcg S/L (WHO), alternate route P/R (slow absorption) dose - 800-1000mcg

Other Add on drug : TRANEXAMIC ACID : iv 1g x 20min

Drug Rx alone is tried for 30min after that

↓

continue Drug Rx + BALLOON TAMPONADE ≤ 500ml NS/RL

↓

Eg: 1. BAKRI BALLOON
2. SENGSTAKEN BLAKEMORE TUBE
3. CONDOM CATHETER

Intrauterine packing using gauze pieces (if unsterile causes SEPSIS, so not done nowdays)

↓

if Still ATONIC UTERUS

Vitals are STABLE

- Do UTERINE ARTERY EMBOLISATION

↓

if Stable

- Do Uterine Compression Sutures

Ⓜ️Ⓒ B-LYNCH Sutures/BRACE Sutures
Others : HAYMAN's SUTURE
CHO SQUARE

→ Still Bleeding →

Vitals are UNSTABLE

Do UTERINE ARTERY LIGATION @ level of Internal OS ; B/L Ligation ; Reduces Pulse Pressure

+

Ligation of ANASTOMOSIS b/w UTERINE ARTERY and OVARIAN ARTERY

↓

Still Bleeding and ATONIC UTERUS

↓

Do HYSTERECTOMY
whenever possible will do
SUPRACERVICAL HYSTERECTOMY
(SUBTOTAL)

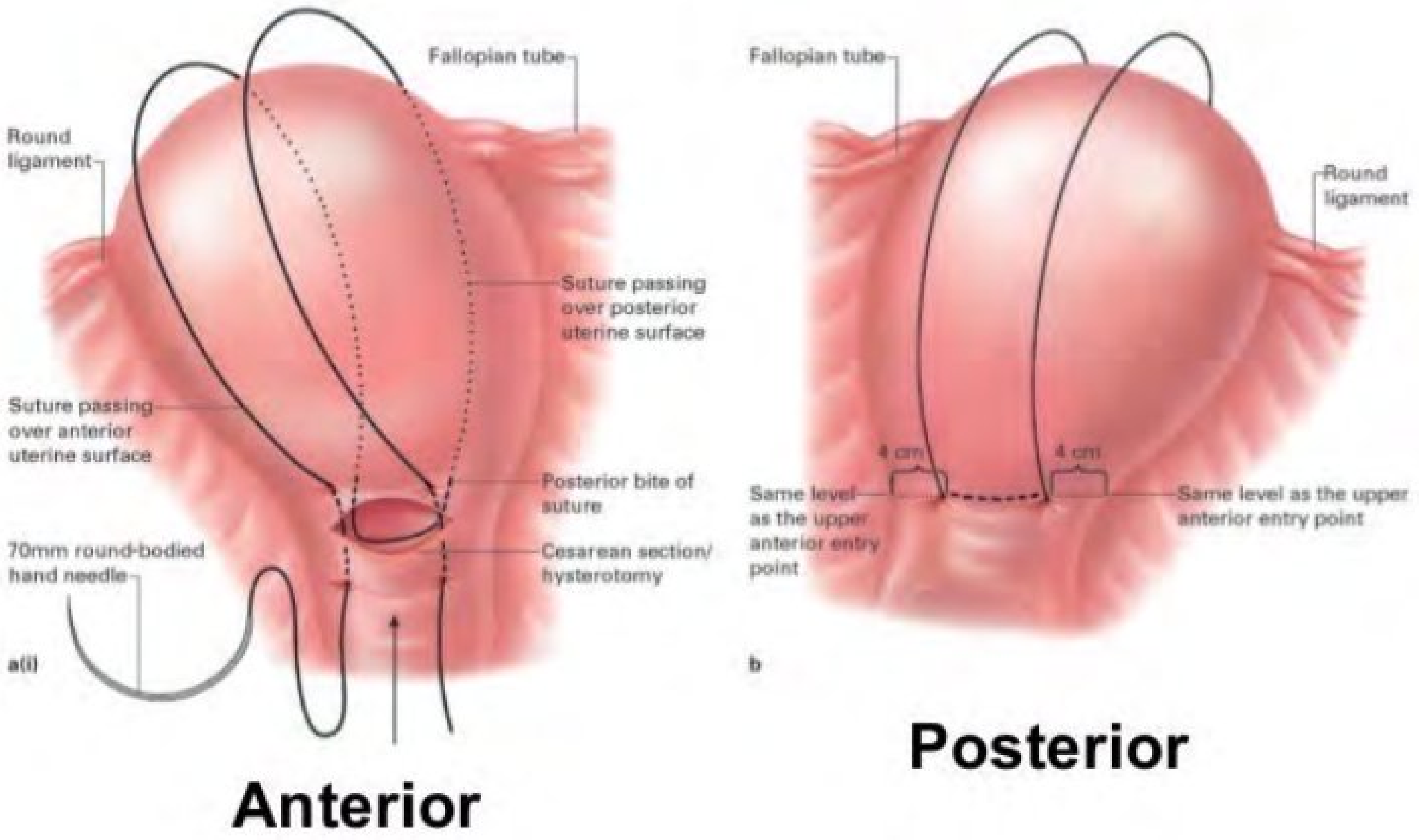
← If Hemostasis can't be achieved ←

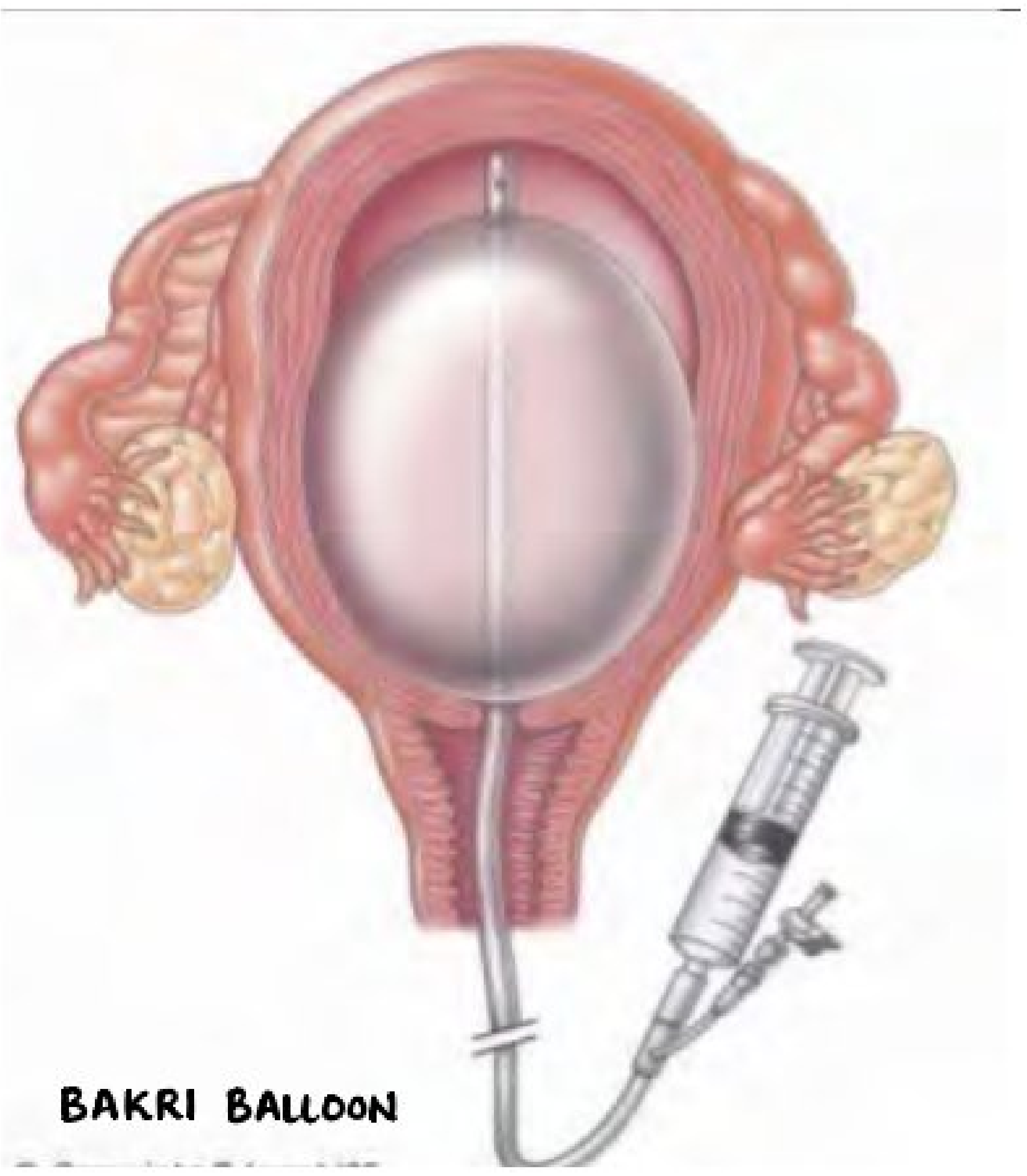
TOTAL HYSTERECTOMY is done
(Uterus and Cervix are removed)

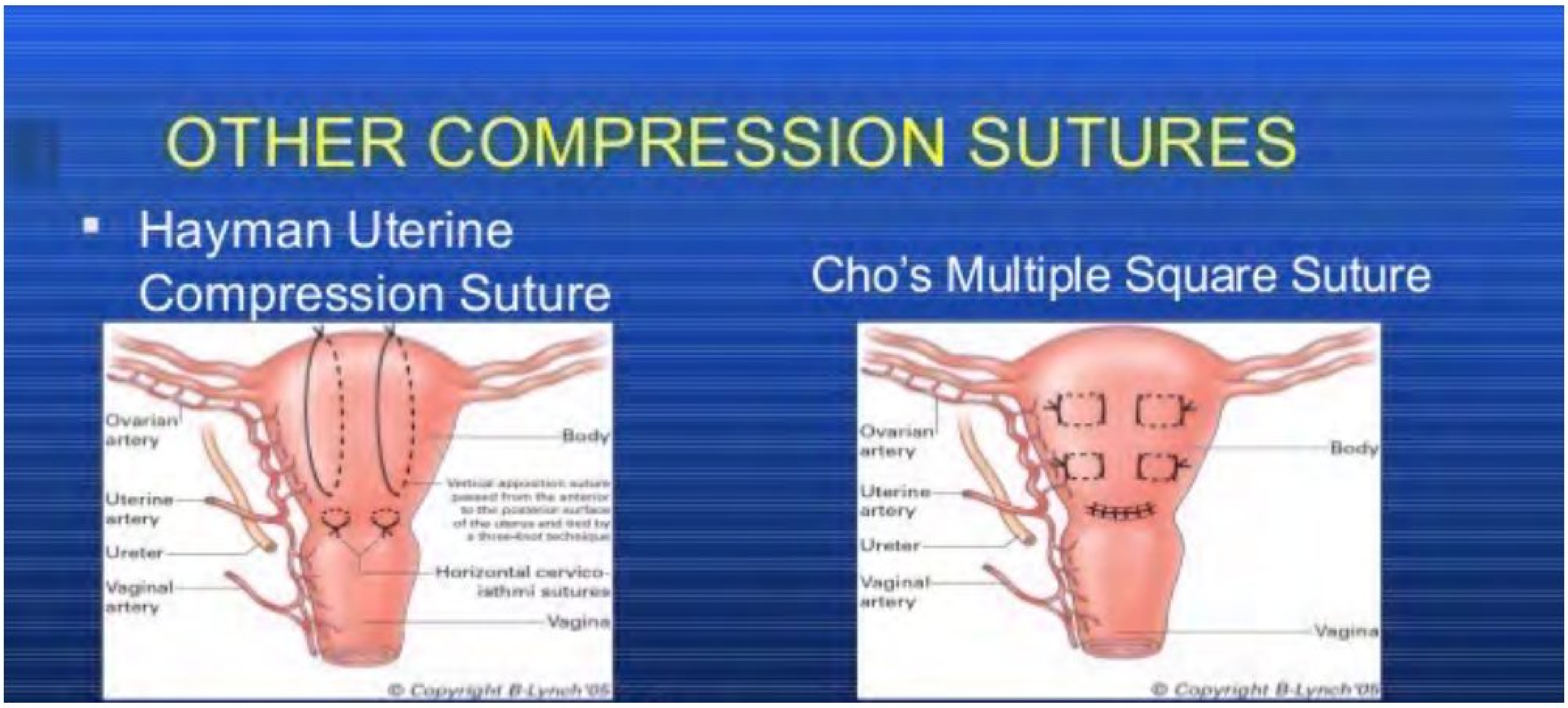
↓

If still bleeding after Hysterectomy : Dx · DIC
(DISSEMINATED INTRAVASCULAR COAGULATION)
aka Consumptive Coagulopathy
FFP is also given

B-Lynch Suture


Round ligament
Fallopian tube
Suture passing over posterior uterine surface
Suture passing over anterior uterine surface
Posterior bite of suture
70mm round-bodied hand needle
Cesarean section/ hysterotomy
a(i)
Anterior
Fallopian tube
Round ligament
4 cm
4 cm
Same level as the upper anterior entry point
Same level as the upper anterior entry point
b
Posterior

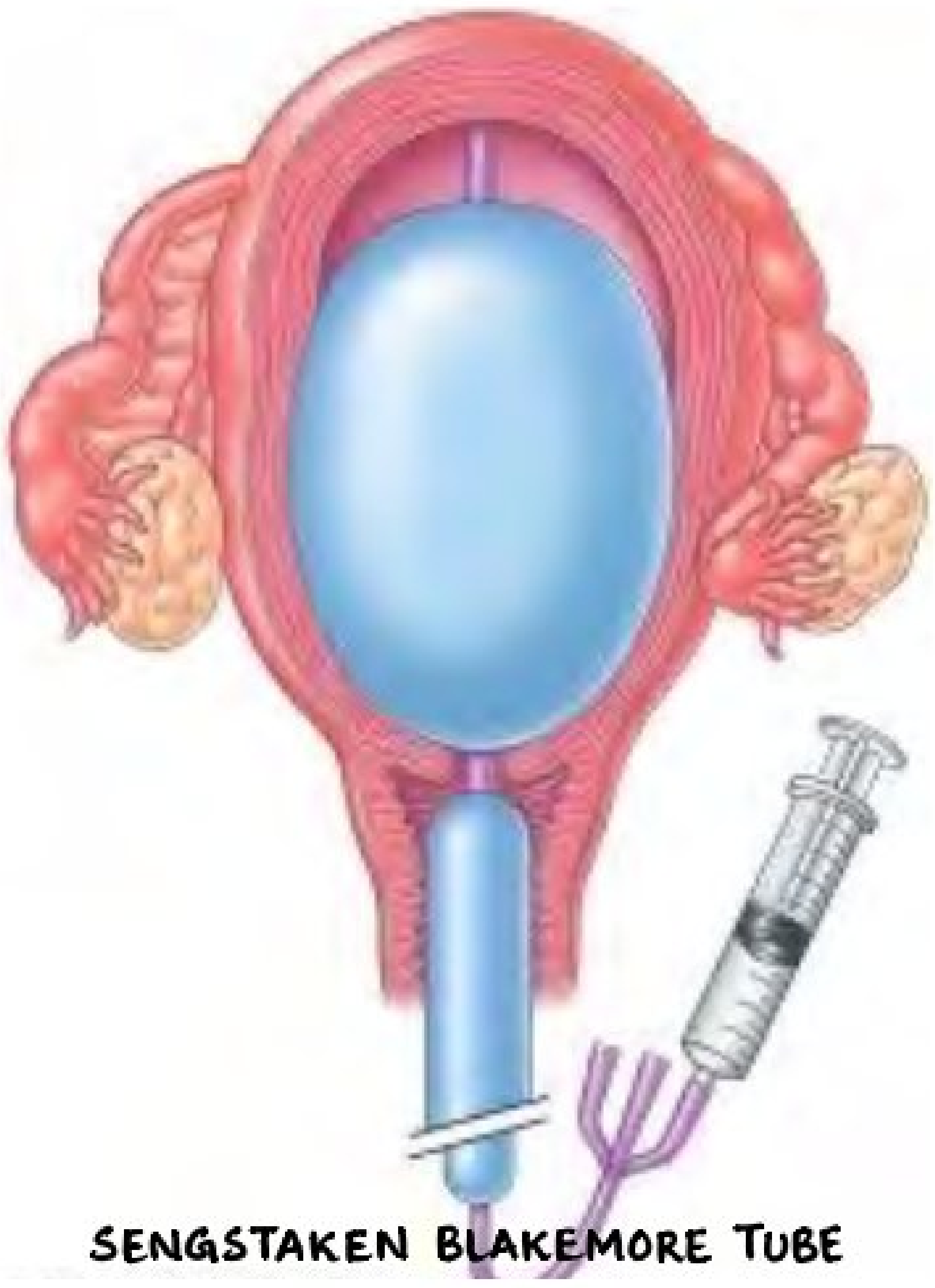

BAKRI BALLOON

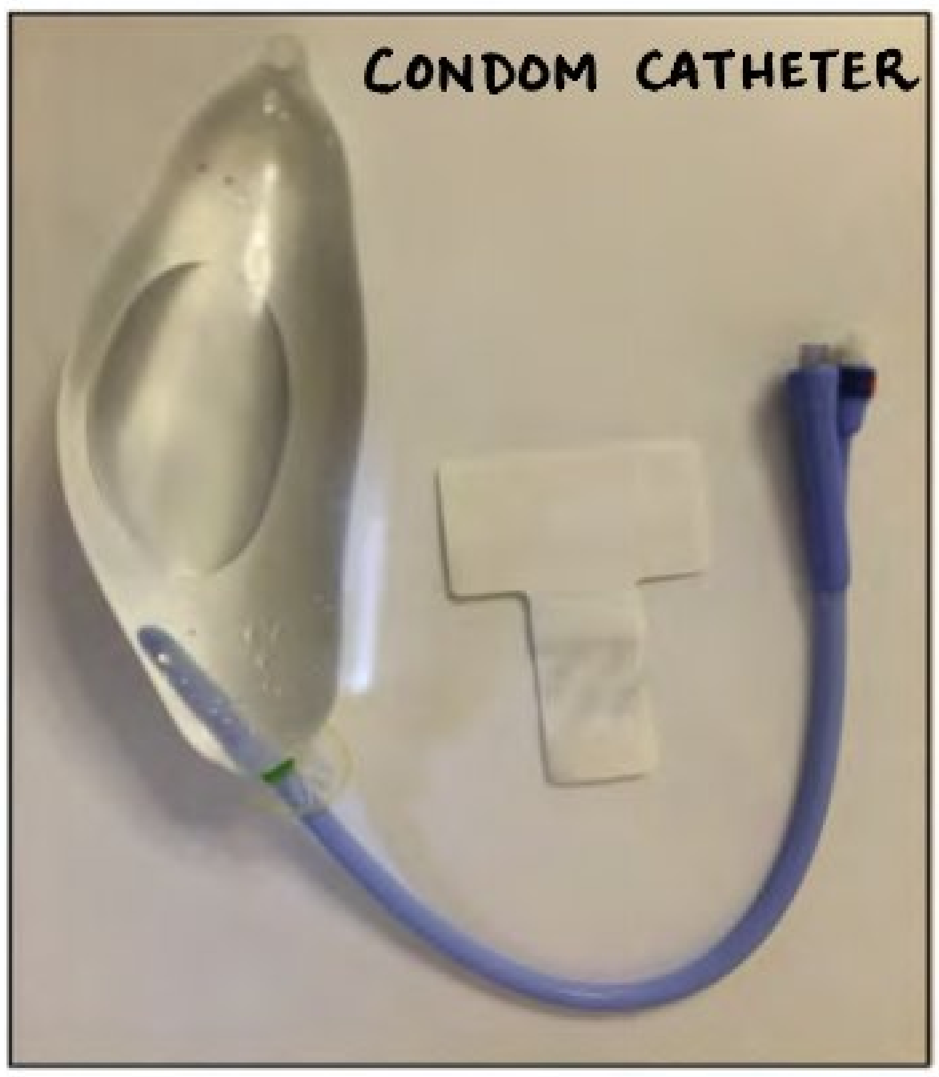


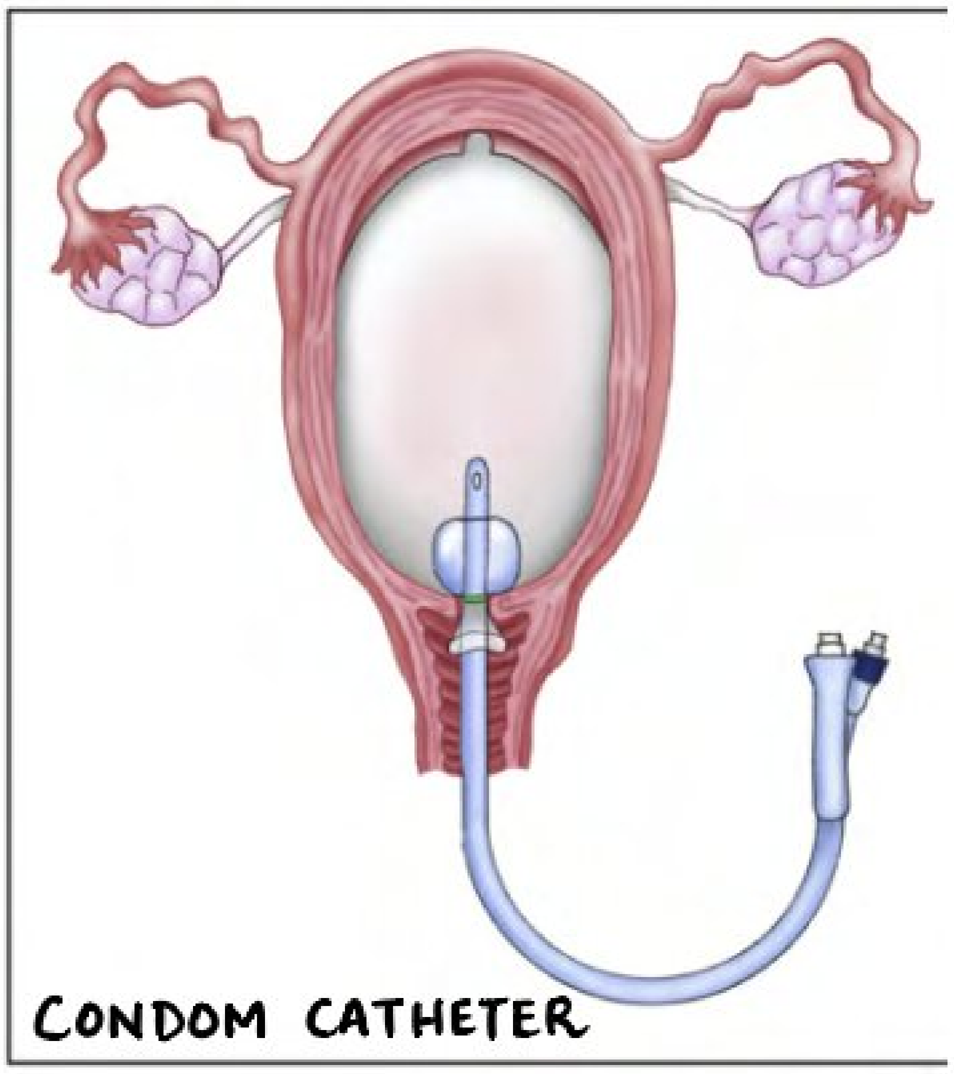
OTHER COMPRESSION SUTURES

- Hayman Uterine Compression Suture

Cho's Multiple Square Suture


SENGSTAKEN BLAKEMORE TUBE


CONDOM CATHETER


CONDOM CATHETER

DIC IN OBSTETRICS : Ⓜ︎Ⓒ cause ABRUPTION

Others: MASSIVE PPH / APH
Septicemia
Intra Uterine Death - Only if Retained >4wks in Utero
↓ most of them spontaneous labour c̄ in 2wks
↓ ↑ in Risk of DIC

Q. 36wks c̄ IUD. Mx? wait and watch
Induction of labour can be done on maternal demand

ANAPHYLACTOID SYNDROME OF PREGNANCY

- Amniotic Fluid Embolism (AFE) can cause PPH
- (MC) occur during labour - when membrane ruptured

↓

1st STAGE of AFE : Sudden development of Breathlessness
↓
Rapidly progress to HYPOTENSION
↓
Rapidly progress to CARDIAC ARREST
↓
COMA
High mortality Rate

2nd STAGE of AFE : DIC presents c̄ Bleeding

CS/Operative deliveries : ↑ Risk of AFE

(MC) AFE - seen after Vaginal delivery

AFE - Rare event ; Dx of Occlusion Clinical Dx

No LAB test for confirmation or refute

PULMONARY VESSEL FINDING - AMNIOTIC FLUID DEBRIS

TRAUMATIC CAUSES OF PPH

TRAUMA TO PERINEUM - 1. Perineal tears
2. Hematoma

1. PERINEAL TEARS

EPISIOTOMY : Surgically planned incisions

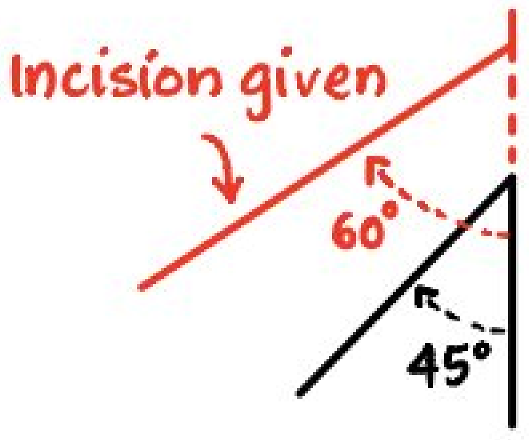
Precautions to prevent perineal tears

1. GOOD Perineal Support
2. Maintain head of the baby in FLEXION to control delivery of HEAD
3. Application of WARM PERINEAL COMPRESSES
4. NO Routine episiotomy

EPISIOTOMY

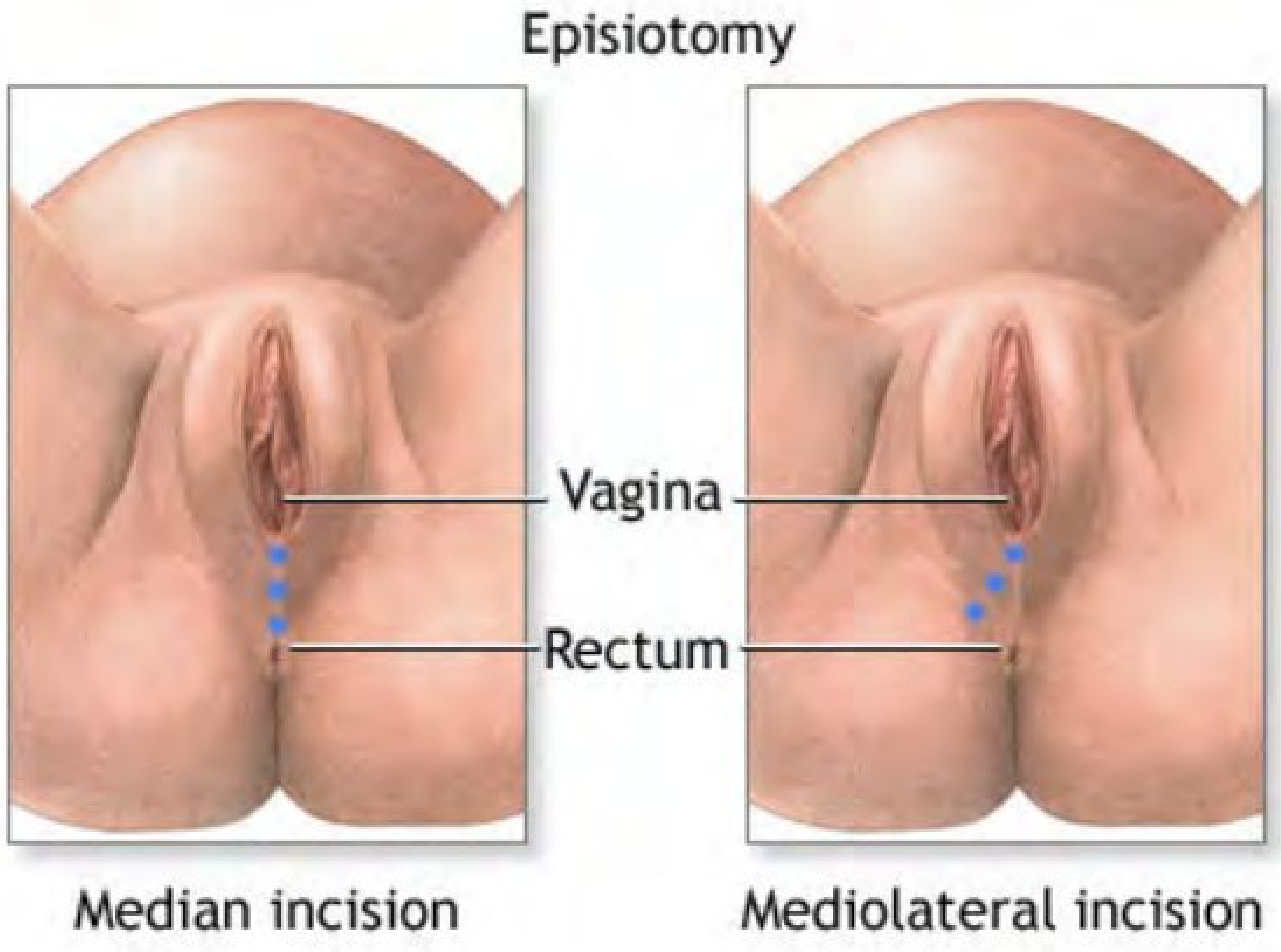
MEDIOLATERAL

- ↑ Blood loss
- ↑ Dyspareunia
- ↑ Breakdown
- Cosmetically POOR
- can be extended



MEDIAN

- if extends it reaches ANAL SHPINCTER; tears in sphincter and anal mucosa leads to RECTAL INCONTINENCE.



EPISIOTOMY is given when perineum is STRETCHED

↳ REPAIR done
1. Vaginal mucosa - CONTINUOUS Suture
2. Perineal muscles - INTERRUPTED Suture
3. Skin - MATTRESS

CLASSIFICATION OF PERINEAL TEARS

1st Degree - if only the skin and/or vaginal mucosa are torn

2nd Degree - 1st + Perineal muscles

3rd Degree - A - <50% of Ext. Anal Sphincter (EAS) is torn

B - >50% "

C - if both EAS + IAS is torn

4th Degree - when RECTAL MUCOSA is torn

3rd and 4th Degree : aka COMPLETE PERINEAL TEAR

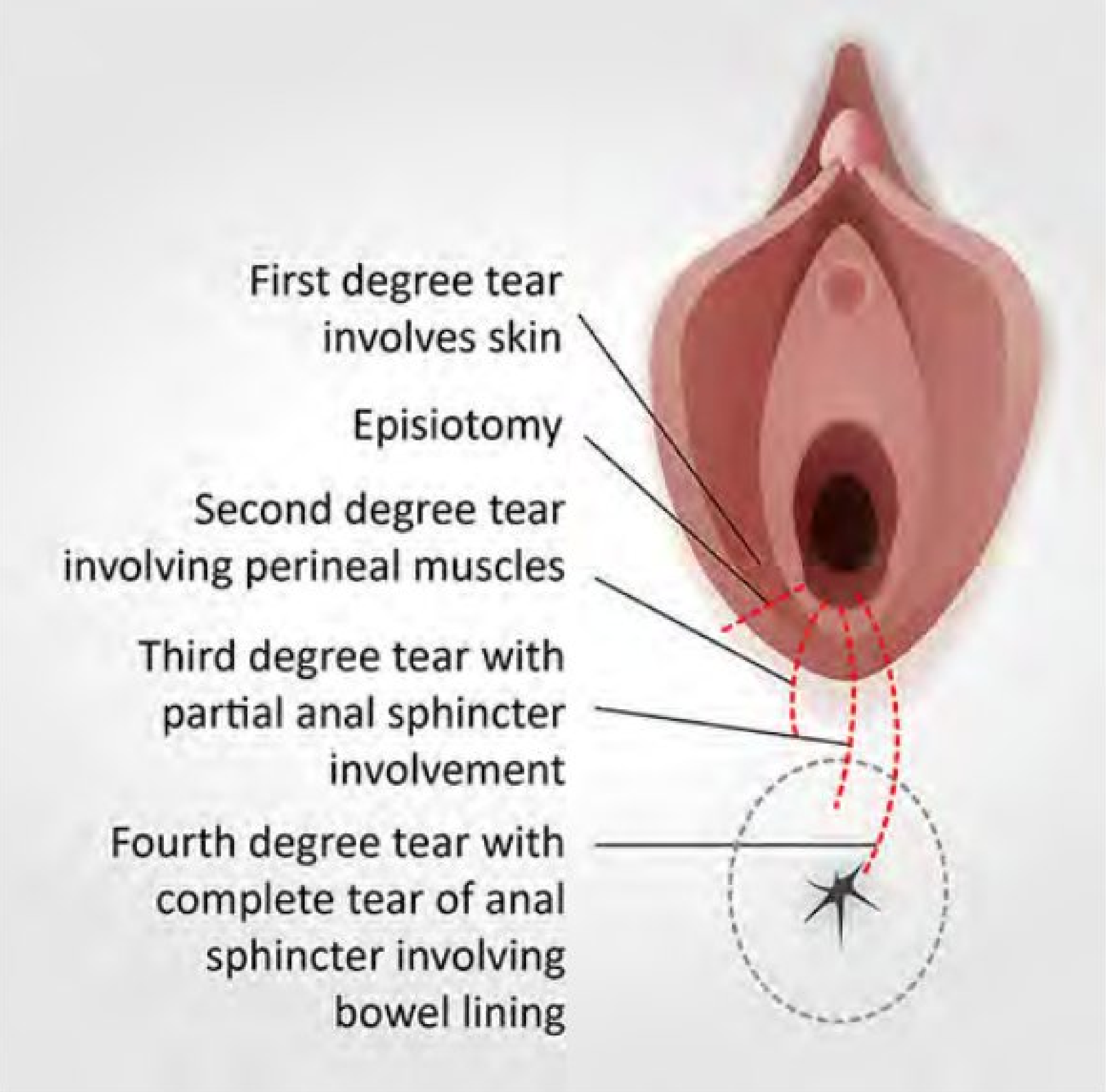
1st and 2nd Degree: Repaired in labour room just like Episiotomy

3rd and 4th Degree : Repaired in OT under Anesthesia

* Repaired c̄ in 24hrs (because of 24hrs

Torns ends will be avascular → NECROSED

1st Structure Repaired - **Rectal mucosa**
↓
IAS
↓
EAS
↓
Rest like EPISIOTOMY


First degree tear involves skin
Episiotomy
Second degree tear involving perineal muscles
Third degree tear with partial anal sphincter involvement
Fourth degree tear with complete tear of anal sphincter involving bowel lining

2. HEMATOMA

1. pain in genital area (MC) presentation
2. Inability to pass urine
3. Shock

Dx by L/E : "BLUISH TENDER SWELLING"

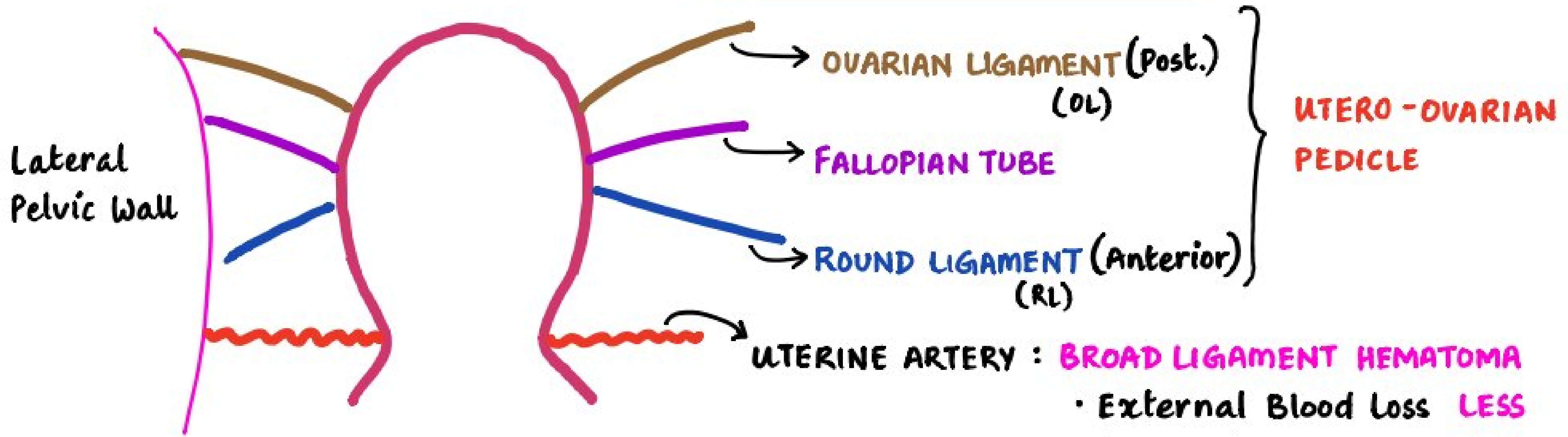
Most hematomas are managed CONSERVATIVELY + ICE PACK

NO ROLE of Antibiotics

INDICATIONS TO OPERATE HEMATOMA

1. UNSTABLE VITALS
2. EXPANDING IN SIZE
3. EXCRUCIATING PAIN (s/i expanding hematoma internally)

Hematoma which presents as SHOCK is **BROAD LIGAMENT HEMATOMA**



UTERINE ARTERY : BROAD LIGAMENT HEMATOMA
- External Blood Loss LESS
- But pt in SHOCK

SUPERO - INFERIOR RELATIONSHIP

Fallopian Tube : Most SUPERIOR

RL and OL : are attached at same level

UTERUS INVERSION

Ⓜⓒ cause IATROGENIC

As soon as Inversion happens ⟶ PAIN ⟶ NEUROGENIC SHOCK ⟶ ATONIC UTERUS + BLEED

↓ converts to

HEMORRHAGIC SHOCK - cause of death

Management : 1. **REPOSIT UTERUS BACK** – Manual Repositioning (**JOHNSON'S METHOD**)

↓

part which comes out LAST has to be pushed in FIRST

↓ if FAILED

Do HYDROSTATIC METHOD / O SULLIVAN'S METHOD

↓ if Fails

OPERATE UNDER G.A

1. HUNTINGTON'S Surgery
2. SPINELLI METHOD
3. HAULTAIN PROCEDURE

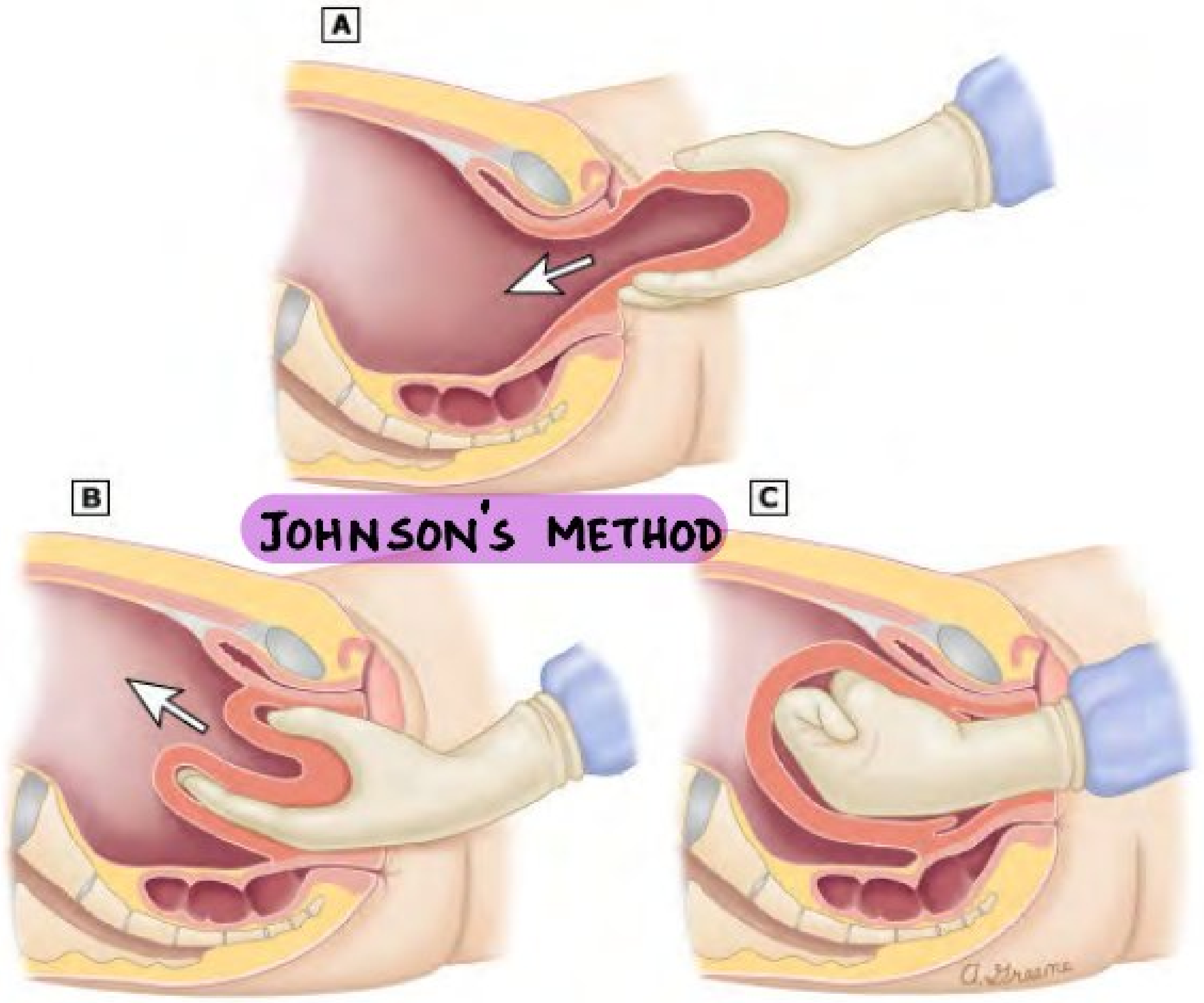
2. **Start OXYTOCIN**

General Anesthesia (G.A)

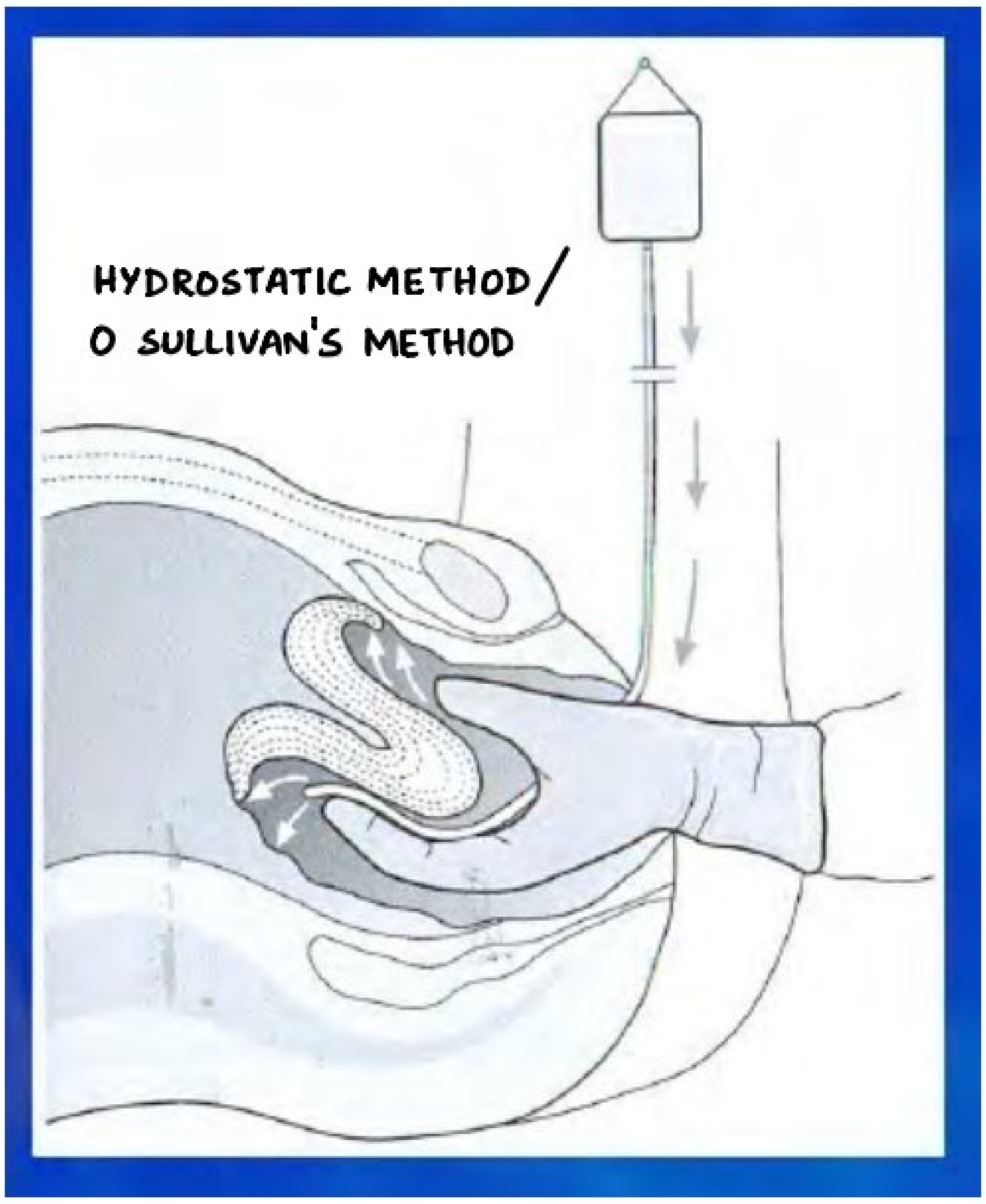
NOT USED in Obstetrics because it can cause INTENSE RELAXATION ⟶ PPH

USED in :

1. SHOCK
2. INTRA UTERINE MANIPULATION
3. SEVERE FETAL DISTRESS



JOHNSON'S METHOD



HYDROSTATIC METHOD / O SULLIVAN'S METHOD

UTERINE RUPTURE

(MC) cause PREVIOUS CAESAREAN

In NON-SCARRED UTERUS RUPTURE

- (MC) cause OBSTRUCTED LABOUR
- Grand Multipara ≥ 5 Times
- MULLERIAN Anomalies

- DRUGS : 1. MISOPROSTOL - if used Antepartum

↓

C/I in Previous C.S for INDUCTION OF LABOUR or AUGMENTATION

2. OXYTOCIN : can also cause rupture.

- So STOP infusion and start i.v fluids Washout OXYTOCIN.

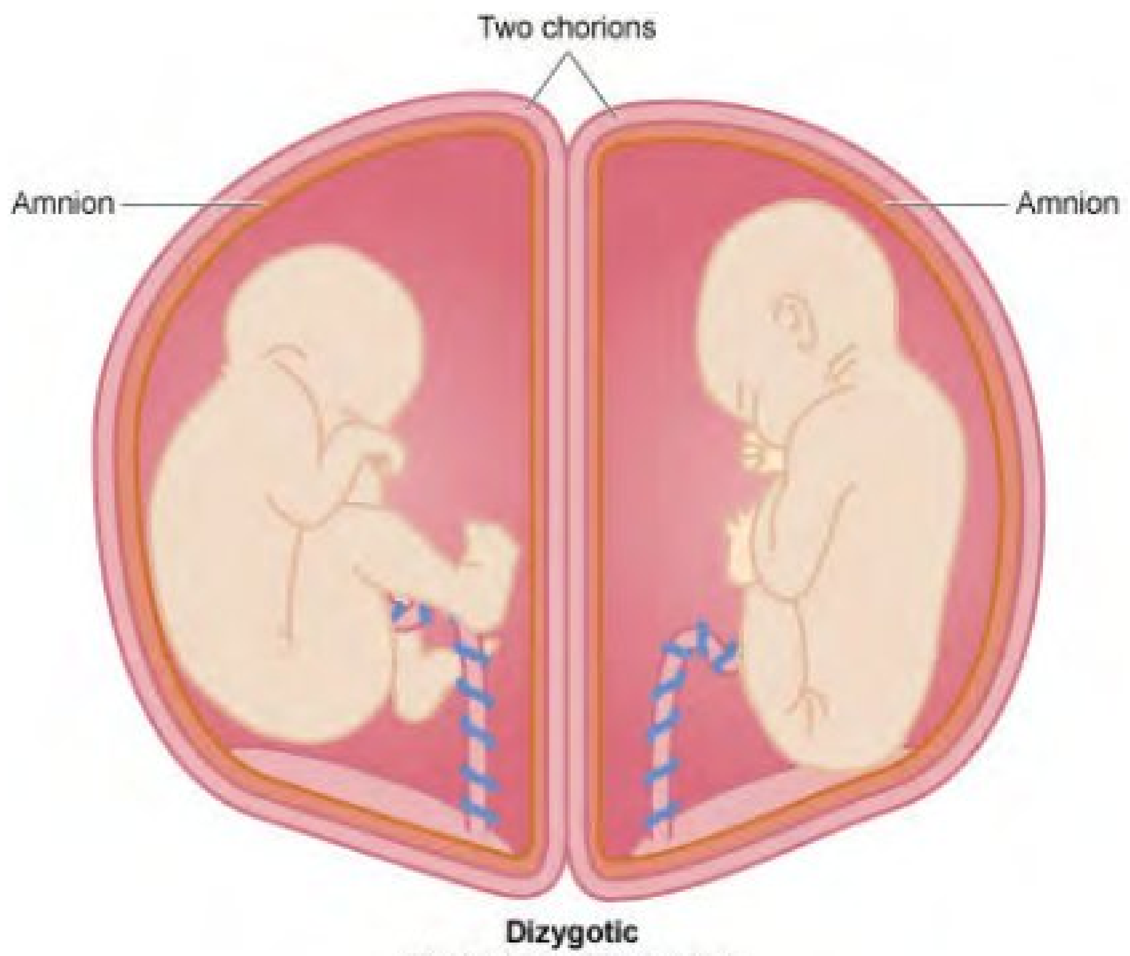
Management : EMERGENCY LAPAROTOMY

↓

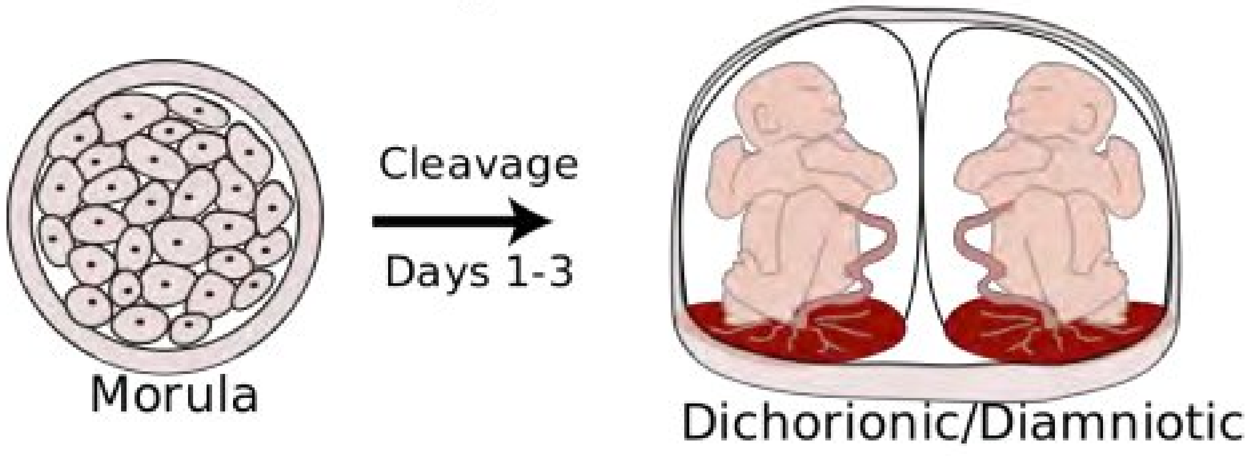
Rupture Uterus - if Hemostasis can't be maintained : HYSTERECTOMY

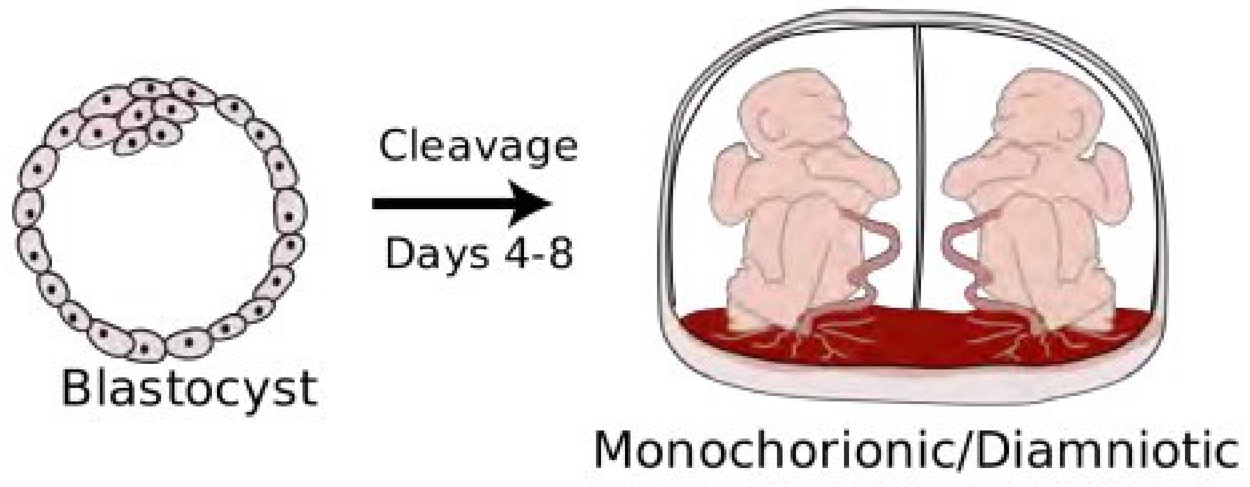
TWIN PREGNANCY

DIZYGOTIC TWINS	MONOZYGOTIC TWINS
• (MC) ~ 70%	• ~ 30%
• Fraternal	• Identical
• Prevalence - Varies -Geographical variation	• Prevalence - Constant
-Family h/o	• TYPES : depends on Time of division
-Race	i) ≤ in 72hrs → DCDA - MORULA STAGE
-ART	ii) b/w 4-8 days → MCDA - BLASTOCYST
• Dichorionic DiAmniotic	iii) b/w 8-12days → MCMA
	iv) >12days → CONJOINED TWINS (SIAMESE Twins) after Embryonic disc



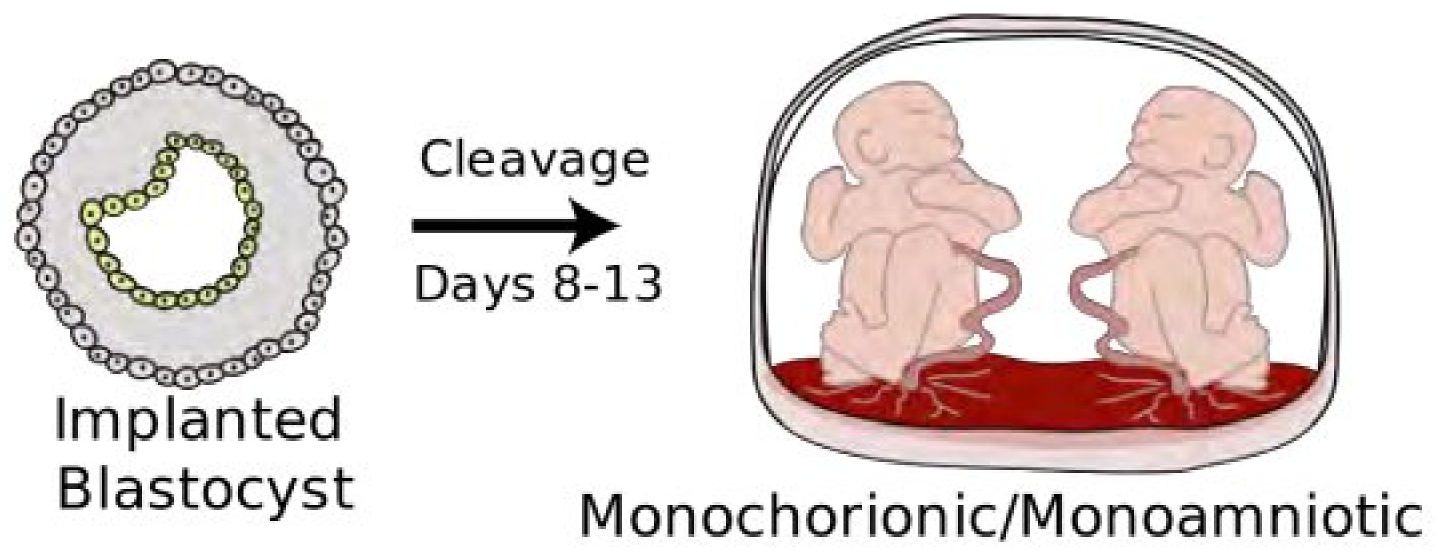
Dizygotic
(Dichorionic, Diamniotic)

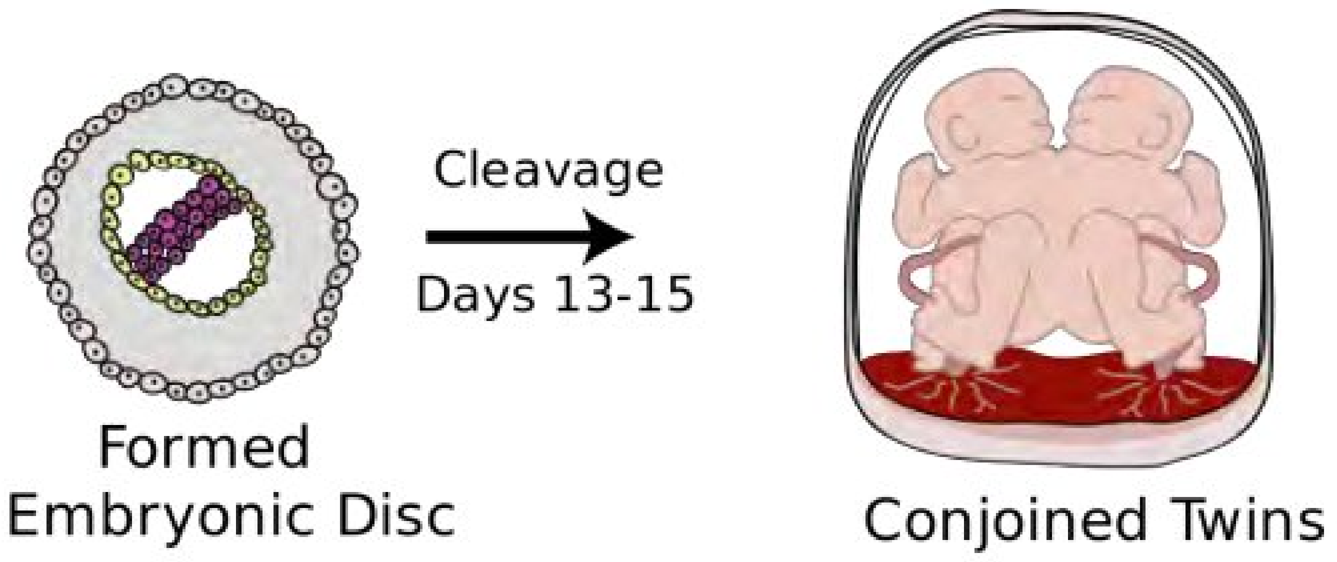




- (MC) Type of TWINS - DCDA
- (MC) Type of Monozygotic twins - MCDA
- Fetus in TWIN Pregnancy is HIGH Risk for all fetal complications except :

1. POST-TERM LABOUR
2. MACROSOMIA



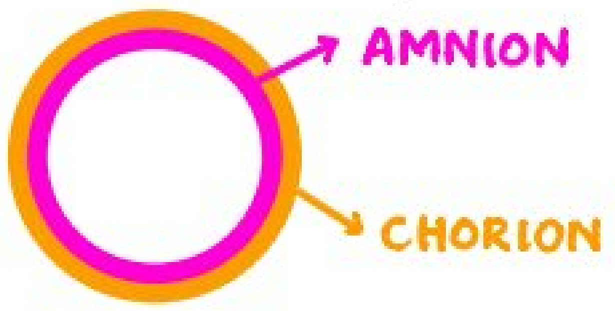


- (MC) fetal complication of Twin Pregnancy

1. Pre-Term (Prematurity) } dependent on Number of Fetuses
2. IUGR

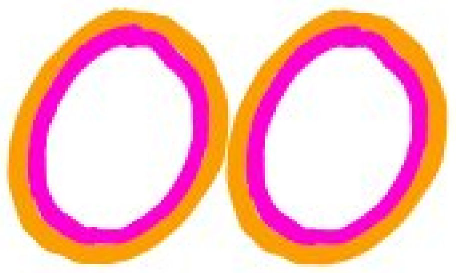
- All fetal complications are more in MONOCHORIONIC TWINS as compared to DICHORIONIC.
- Ridge present b/w 2 cords? → If NO → MAMC

↓ if YES, then?



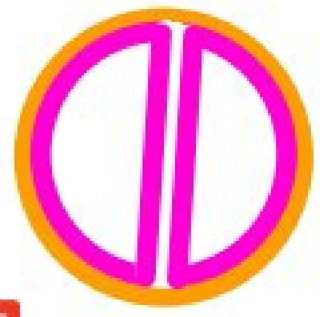
THICK Ridge

- DCDA
vascular connection not present b/w 2 cords



THIN Ridge

- MCDA : if vascular connection b/w 2 cords present

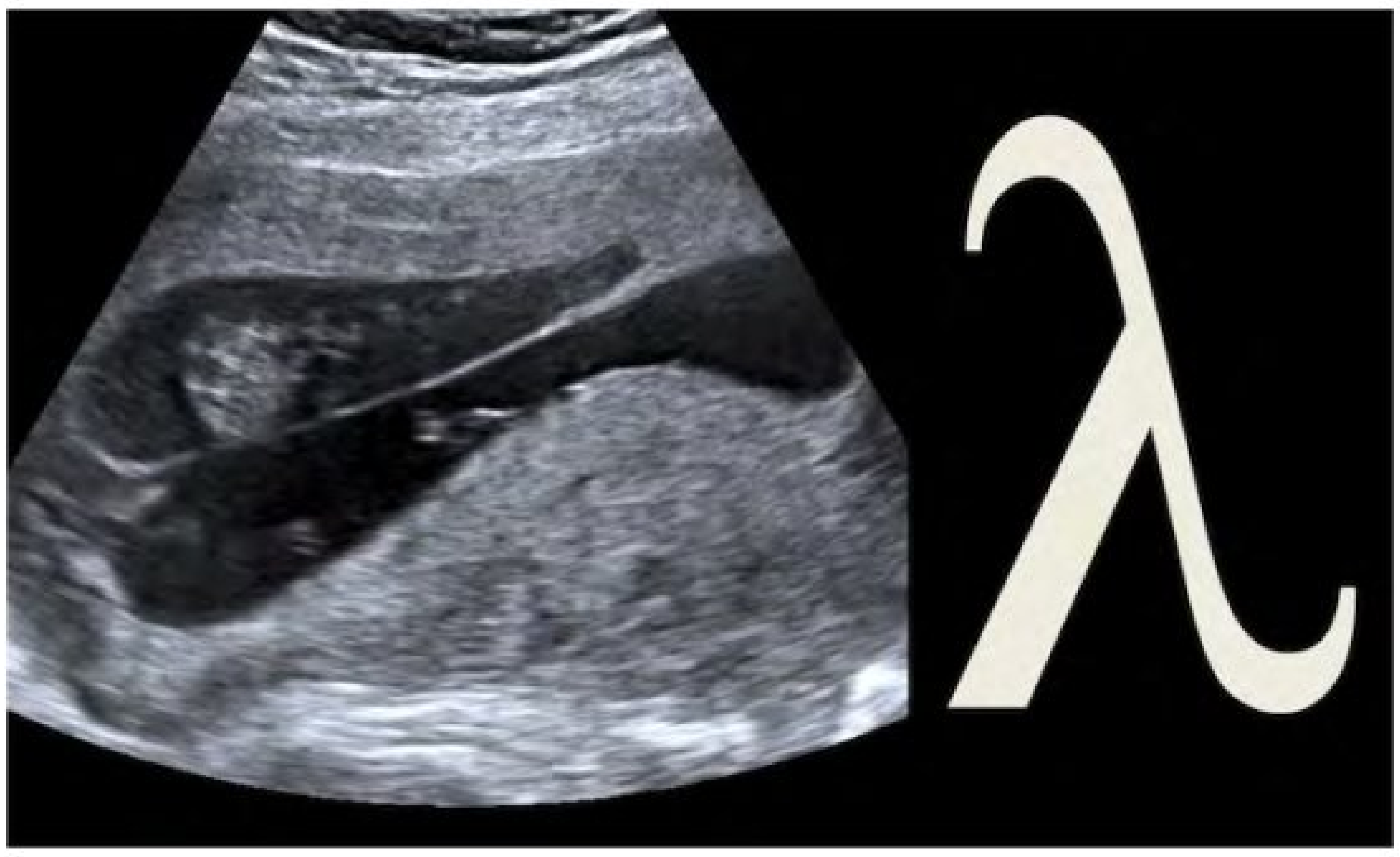


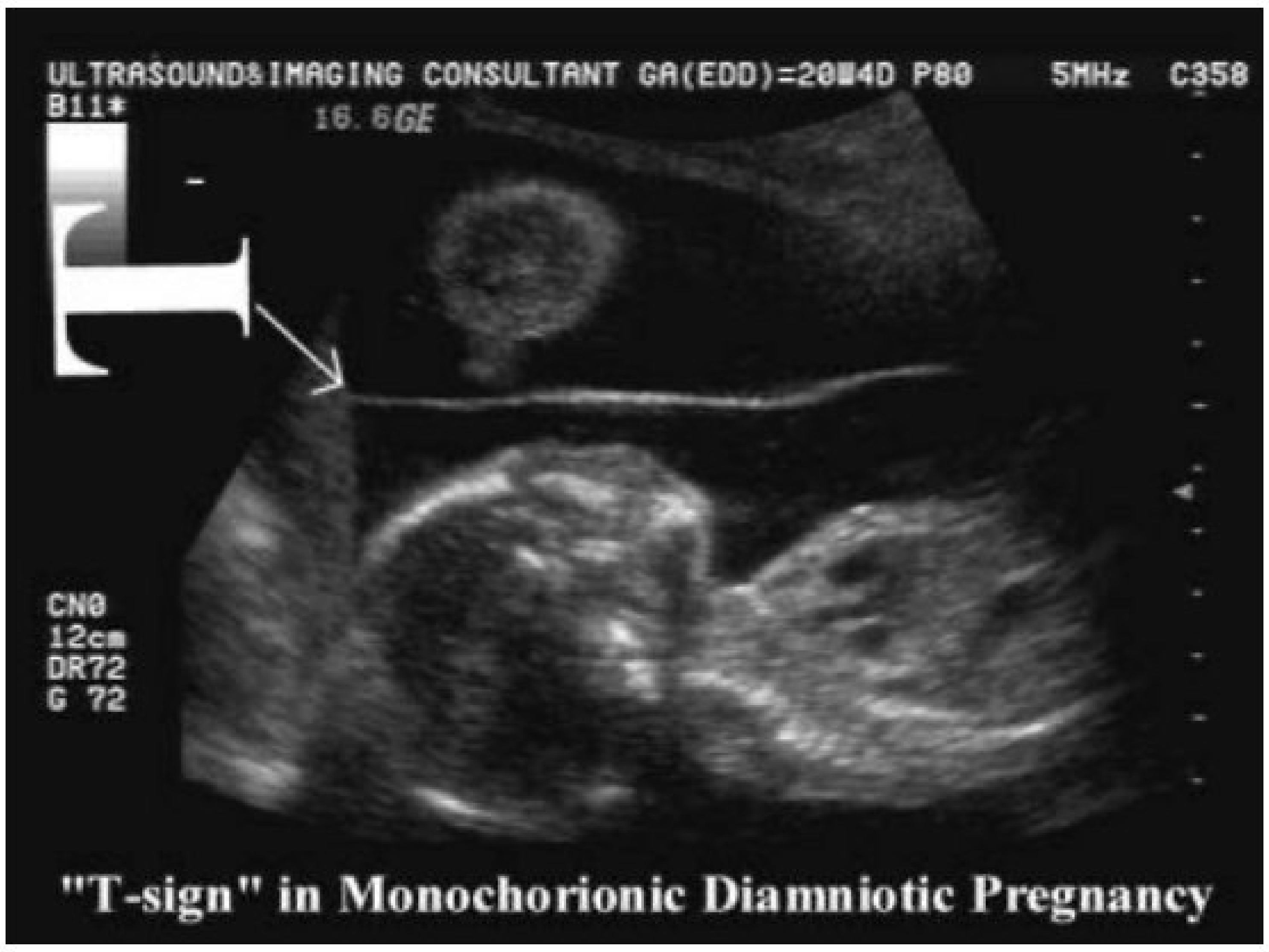
MARKERS OF DICHORIONICITY [USG]

1. If we see 2 separate placenta - GOOD MARKER
2. Opposite Sex Twins - always DICHORIONIC → Good marker
3. TWIN PEAK Sign [LAMBDA SIGN] : Ideal time to look for chorionicity - 10-14wks
4. 4 Layers in dividing membrane
5. If the dividing membrane is THICK [≥2mm]
6. 'T' Sign / Inverted T Sign → S/o MONOCHORIONIC TWINS

Monochorionic is at high Risk than Dichorionic : Characteristic Complication

1. TWIN TWIN TRANSFUSION Synd (TTTS)
2. TWIN REVERSED ARTERIAL Perfusion (TRAP)
3. TWIN ANEMIA POLYCYTHEMIA Sequence (TAPS)
4. Selective IUGR





"T-sign" in Monochorionic Diamniotic Pregnancy

TWIN - TWIN TRANSFUSION SYNDROME

Monochorionic
DA >> MA

- cause of DA : vascular connections / vascular anastomosis (Deep artery → Deep vein)

UNEQUAL BLOOD FLOW
↓

Deep Vein (Recipient twin) → ↑ Blood volume → Kidney → POLYURIA → POLYHYRAMINOS

Deep Artery (Donor twin) → ↓ Blood volume → Kidney → OLIGURIA → OLIGOHYDRAMINOS

Diagnosis on USG → Simultaneous presence of Poly / Oligo "STUCK TWIN" → Oligo baby

STAGING : QUINTERO

Stage 1: Polyhydraminos / oligohydraminos + Bladder of Oligo twin is visible → DOPPLER STUDY - Ⓝ

Stage 2: Poly / Oligo + Bladder is not visible + Doppler Ⓝ

Stage 3: Poly / Oligo + Bladder is not visible + Doppler AbⓃ

Stage 4: Either One / Both Fetus - develops Hydrops fetalis

Stage 5: Either One / Both die

Management: 1. Fetoscopic LASER Ablation of Vascular Anastomosis

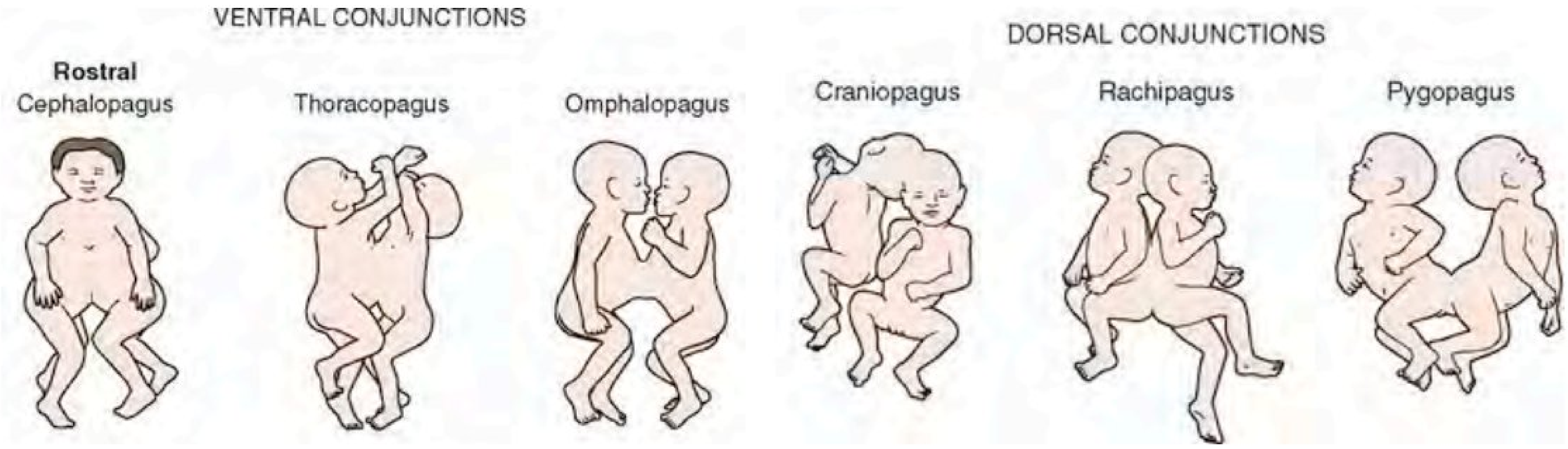
Most characteristic complication of Monoamniotic twins - CORD ENTANGLEMENT - Formation of KNOT

It can cause IUD

if MONOAMNIOTIC → deliver by C·S → 32-34 wks

SIAMESE TWINS - eg. of Monoamniotic
↳ (MC) Type of Conjoined Twins → PARAPAGUS > THORACOPAGUS

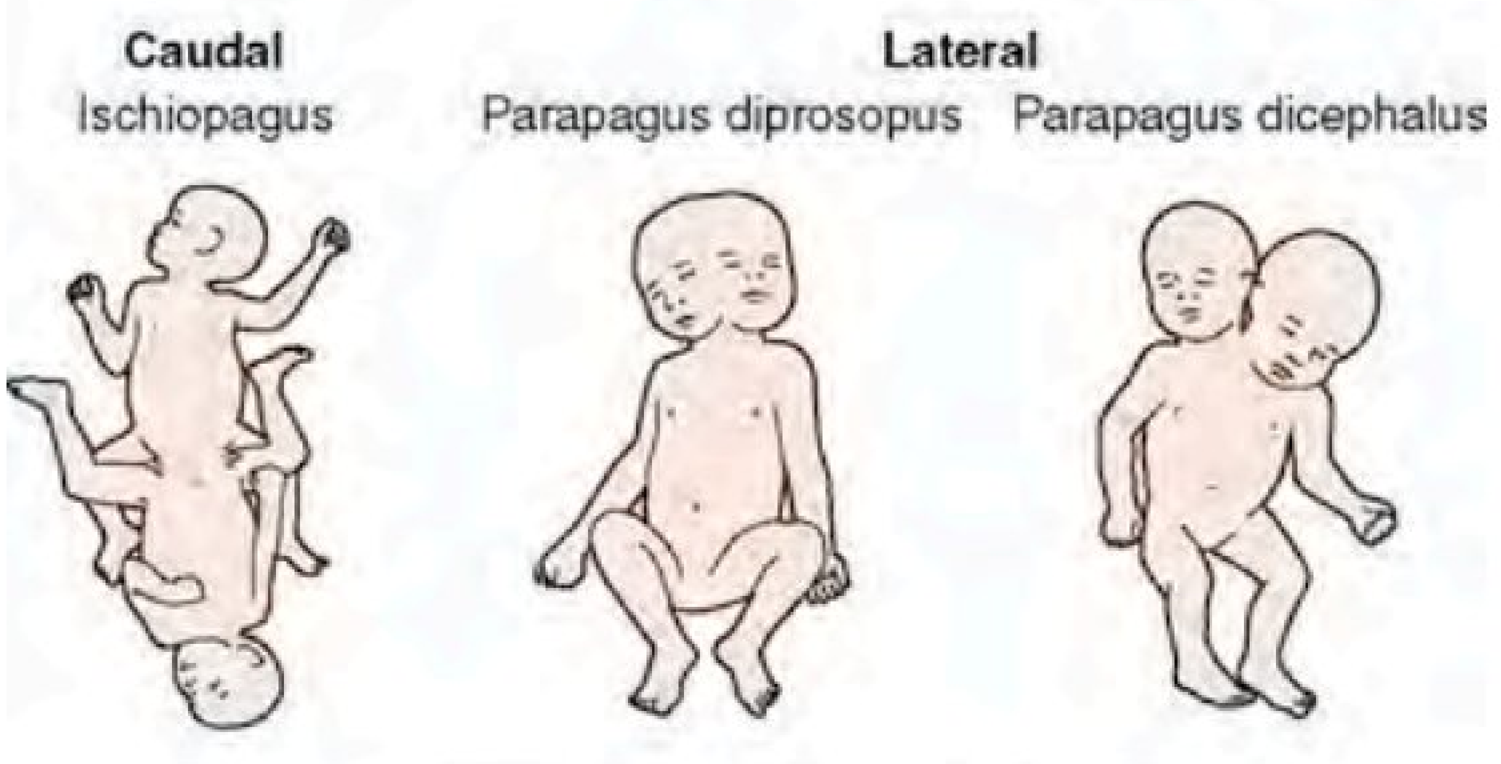
(LC) Type - RACHIPAGUS > CRANIOPAGUS



If twins are DIAMNIOTIC : depends on LIE of 1st TWIN
(first fetus to enter pelvis)

(MC) Type in Labour - VERTEX - VERTEX

2nd (MC) Type - VERTEX - BREECH



if 1st TWIN - Vertex → V·D

if 1st TWIN - Non-Vertex → C·S

TRANSVERSE LIE
↓
C·S

BREECH : C·S Preferred, V·D not C/I

TWIN INTERLOCK
1st Twin Breech - after coming head of 1st twin gets locked c̄ head of 2nd twin which is VERTEX.

1st TWIN - vertex → Delivered → 2nd TWIN → If BREECH → Assisted Breech V·D

↳ If TRANSVERSE → IPV [INTERNAL PODALIC VERSION]
- Intra uterine manipulation
- done in OT under Anesthesia
- Breech extraction.

FETAL REDUCTION

- in IVF ; Embryos → in Uterus
  ↳ QUADRUPLETS → Reduce the no. of fetus
- its an USG Procedure - 10-13wk → Inj. KCL [Cardiotoxic] to baby → Die and gets absorbed.

PREVIOUS C·S

1. Repeat C·S

2. VBAC (Vaginal Birth After C·S) aka Trial of Scar

1. REPEAT C·S

Absolute Indications

i) Previous C·S – Classical C·S – Scar on the upper segment of uterus - weak scar
Risk of Rupture : 4-9%
Classical in Prev. Pregnancy → Repeat C·S → Give LUS Transverse incision

ii) H/o Rupture uterus

iii) ≥ 3 Prev. LSCS

Q. $G_2P_1L_1$ c̄ prev. CS ↷
look for CPD ⊕
Current Preg. → No VBAC
Prev. Preg. → VBAC

iv) Surgery on uterus such as HYSTEROTOMY

- incision scar in upper segment
- done in 2nd TM abortion done following failure of all methods

v) CPD in Current Pregnancy: Trial of Labour is done early only in non-scarred uterus

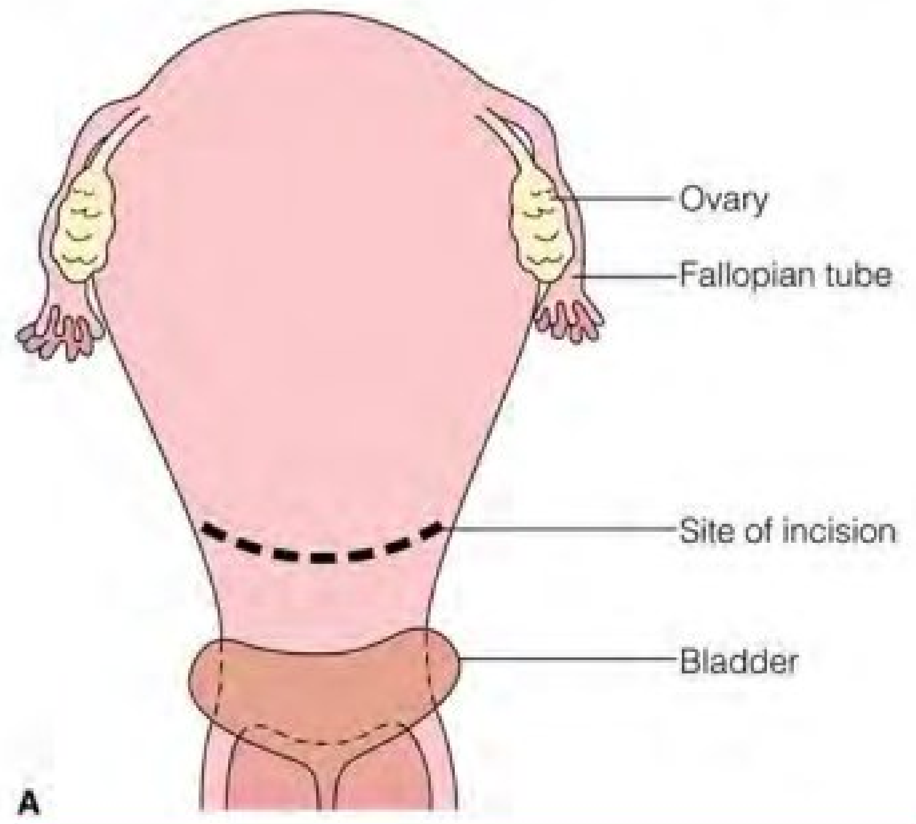
vi) Vaginal delivery NOT possible eg: i) CPD ii) Placenta praevia

Other Types of C·S

1. Pfannensteil incision (or) Low Transverse Incision

Ⓜⓒ used - LSCS ; name : KERR's Incision
Strong Scar ;

Risk of Rupture - 0.2 - 0.9 %



2. Vertical Incision (small)
   - LUS ; Risk of Rupture 1-7%

Relative Indications

i) Prev. C·S c̄ Breech presentation

ii) Prev. C·S c̄ Macrosomia

iii) Prev. C·S c̄ Post-Term Pregnancy

CLASSICAL C·S in MODERN OBSTETRICS

1. C·A Cervix
2. Ds adhesions b/w LUS and Bladder
3. Vesicovaginal fistula
4. Post mortem

TRIAL OF VBAC / OF SCAR

- Successful will deliver vaginaly
  - ↓ Prognostic marker : Prev. h/o VD
- Impending Rupture
- Rupture
  - (MC) cause Prev. CS

IMPENDING RUPTURE

Signs and Symptoms

1. (MC) / most consistent / First Sign - Pattern
   - Non-Reassuring FHR
   - 1st Tachycardia } Fetal
   - (MC) Bradycardia } Fetal
2. Maternal Tachycardia } Become significant if they are seen with Non-Reassuring FHR
3. Scar Tenderness } Become significant if they are seen with Non-Reassuring FHR

Management

- Emergency C.S → Scar dehiscence - when all layers of myometrium give way but serosa is intact
  - it is an Intra-operative clinical Dx

RUPTURE

- all layers of myometrium give way including the Serosa → Fetus is expelled into Abdomen → loses vascular connection → FETAL DEATH

Signs and Symptoms:

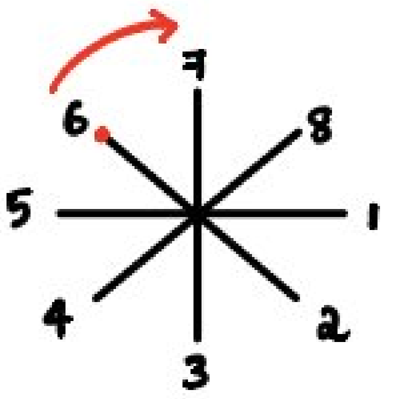
1. Maternal Tachycardia
2. Hypotension ±
3. Fetal HR - severe distress Absent
4. Fetal parts - felt more Superficial or easily palpable
5. Uterus Scar contour → LOST
6. Sudden Stoppage of Uterus contraction
7. Fresh Bleeding P/v
8. Hematuria
9. On P/v - loss of Station (head will recede back)
   ↳ Best sign of Rupture uterus

Management : Emergency Laparotomy → 1st Repair $\xrightarrow[\text{hemostasis}]{\text{if failed}}$ HYSTERECTOMY

3rd Outcome : In ANTHROPOID Pelvis - Direct OP

Vaginal delivery possible
Face to Pubis → C·S

Persistent OP → C·S



MALPRESENTATIONS

- (MC) BREECH

BREECH → High Risk of Gross Congenital Anomalies ~ 6%

↓

(MC) cause Prematurity

@ 28 wks – 25%
@ 32 wks – 7-11%
@ TERM – 3%

Recurrence Risk of Breech – 10%

- Risk Factors for Malpresentations

1. Ab(N) Pelvis	5. Multifetal Pregnancy
2. Multiparity	6. Polyhydraminos / Oligohydraminos
3. Prematurity	7. Fetal Malformation
4. Placenta Praevia	8. Uterine malformations

TYPES OF BREECH

1. EXTENDED BREECH / FRANK BREECH - (MC) ~ 65%, on P/v : heel, feet not felt; (MC) in PRIMI

2. COMPLETE BREECH / FLEXED BREECH -
- Feet close to buttocks
- On P/v : Heel of the feet felt
- (LC) Type of Breech

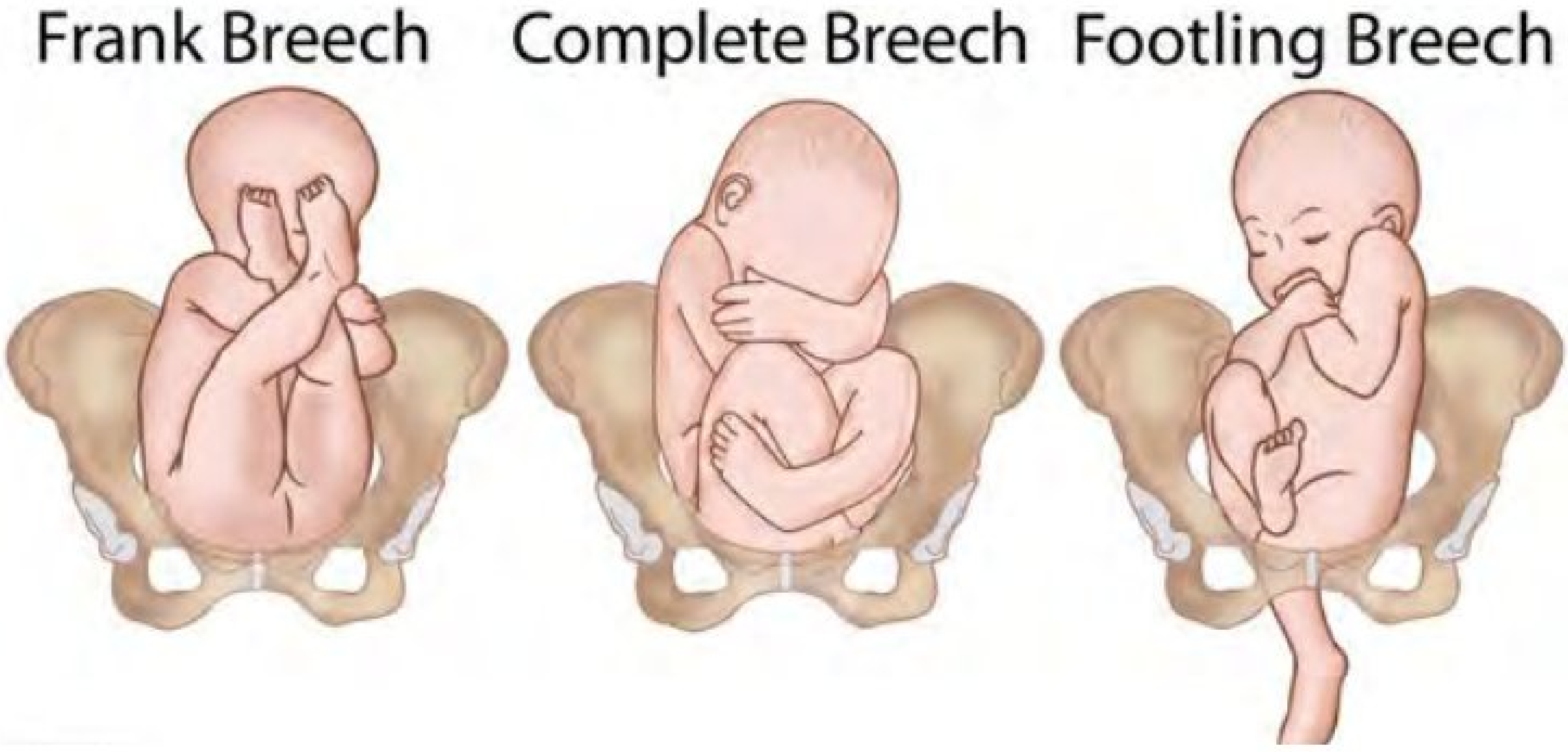
↳ (MC) in MULTIPARA

3. INCOMPLETE / FOOTLING BREECH → SINGLE / DOUBLE

- Feet comes out
- Hip and Knee joint partially Extended
- UMBILICAL CORD can fall out

- Highest Risk of CORD PROLAPSE → in BREECH: INCOMPLETE
  - ↗ in general : TRANSVERSE LIE
- Least Risk of CORD PROLAPSE → in general : VERTEX Presentation
  - ↳ in BREECH : FRANK

- **FOOTLING Breech - Absolute Indication for C·S**


Frank Breech Complete Breech Footling Breech

INDICATIONS FOR C·S IN BREECH

1. RELATIVE INDICATIONS

- CS is preferred ; V·D is not C/I
- Cervix open (PARTIALLY) – 5cm dilated
  - Buttocks
  - After coming → Head → can get stuck → Suffer from HYPOXIA cause
    1. CEREBRAL PALSY
    2. DEATH – if severe enough

i) PRIMI c̄ BREECH

ii) Previous C·S c̄ Breech

iii) HYDROCEPHALUS c̄ BREECH
↳ After C·S → VP Shunt done

iv) Macrosomia (≥ 4kg)

v) Pre-Term (viable Breech)

2. ABSOLUTE INDICATIONS

: i) FOOTLING BREECH

ii) STARGAZER Baby : Neck extended

Primi 37wks → Presents c̄ BREECH
↓
Try

EXTERNAL CEPHALIC VERSION (ECV)

- Tried in all non-cephalic presentation (eg: Transverse lie) provided requirements are met.

• Requirements on USG

i) Frank and Complete Breech

ii) Adequate Liquor

iii) Membranes Intact

iv) SINGLETON Pregnancy

v) FHR – Ⓝ

vi) VD possible

vii) Latent phase

• CONTRAINDICATIONS

i) Footling

ii) Oligohydraminos

iii) Ruptured Membrane

iv) TWINS / MULTIFETAL

v) Previous C·S

vi) Fetal distress

vii) Placenta praevia

viii) ACTIVE LABOUR

- If Requirements are met ECV done

1. Ideal Time ~ 36-37wks (not done
   - at 38wks – ↑ Weight
   - at 35wks – Can REVERSE, can go into PRETERM Labour

2. under continuous Fetal monitoring - if fetal distress - REVERSION → Take for C·S

3. under TOCOLYSIS

Requirements NOT met for ECV → Indications for C·S
↓

ASSISTED BREECH VAGINAL DELIVERY

1. LOVSET'S MANEUVER : if arms are EXTENDED

2. PINARD'S MANEUVER : if legs are EXTENDED

3. BURN'S MARSHALL MANEUVER

- Spine anterior
- Baby delivered by FLEXION
- 180° towards Maternal Abdomen
- for after coming head

4. MAURICEAU - SMELLIE - VIET (MSV MANEUVER)

- for after coming head
- Spine anterior
- Malar flexion and Shoulder traction

5. PRAGUE'S MANEUVER

- Baby spine posterior/dorso posterior
- delivery extension of head
- for after coming head

6. DUHRSEN'S INCISION

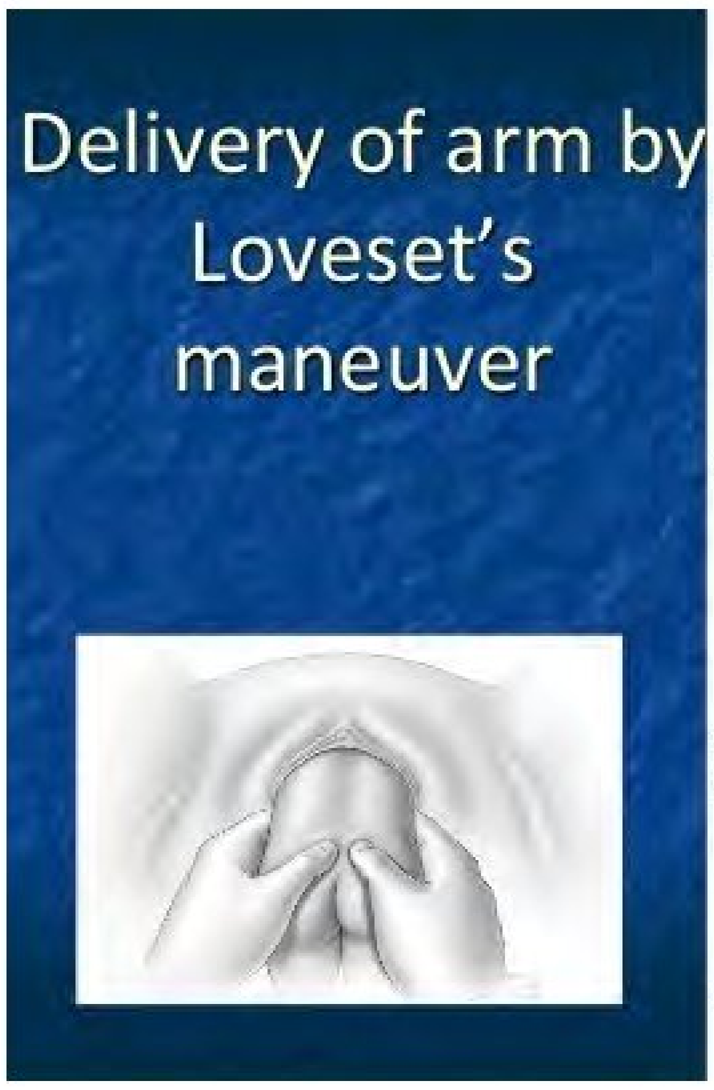
- 2 small incisions on Cervix
- 2'o' clock and 10'o' clock
- Only done for PRE-TERM Babies

7. Delivery of aftercoming head by PIPER's Forceps : Babies body rests on PERINEAL curve of Forceps

8. ZAVANELLI MANEUVER

- Last Resort ; baby pushed back and do C·S

Ⓜⓒ cause of Fetal death in BREECH delivery = INTRACRANIAL Hemorrhage


Delivery of arm by Loveset's maneuver

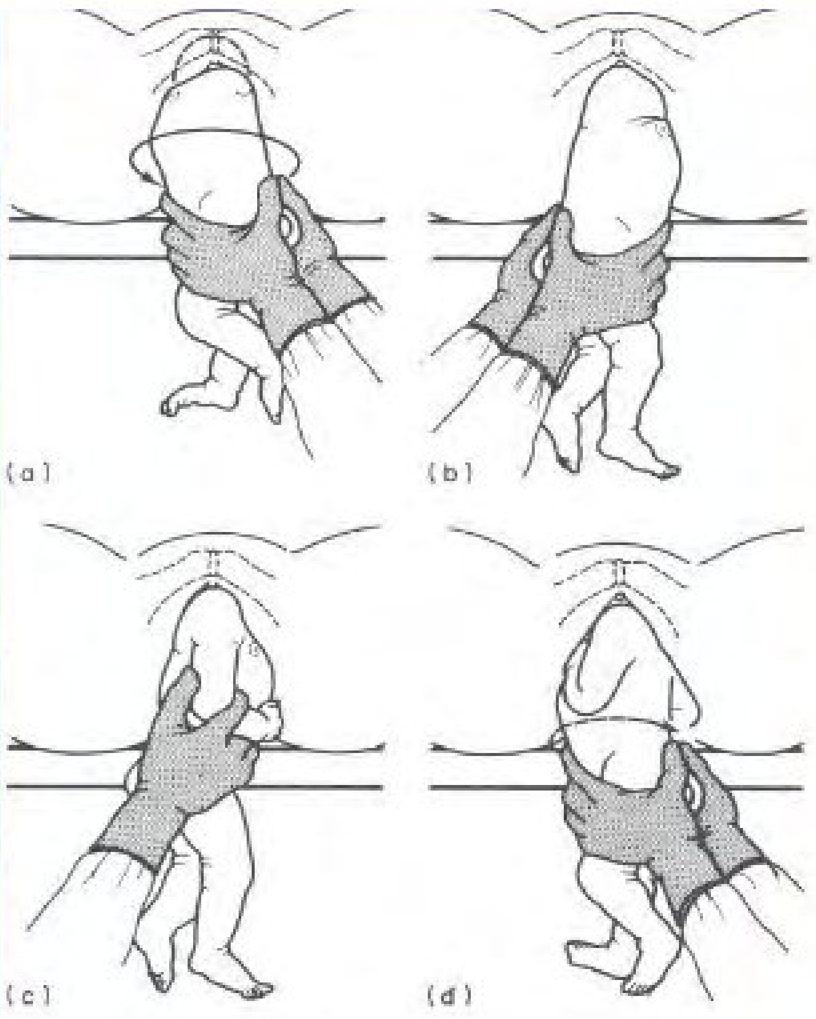
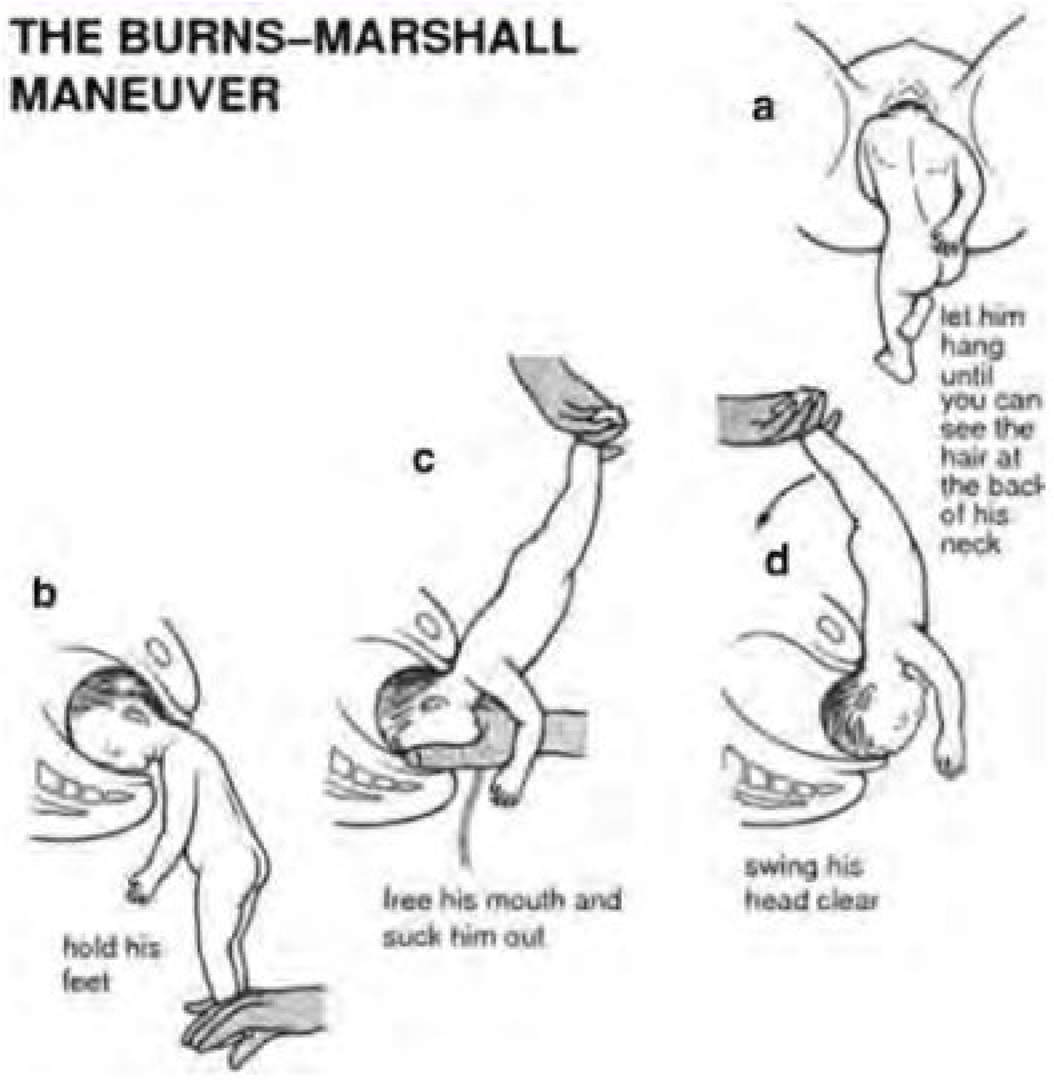
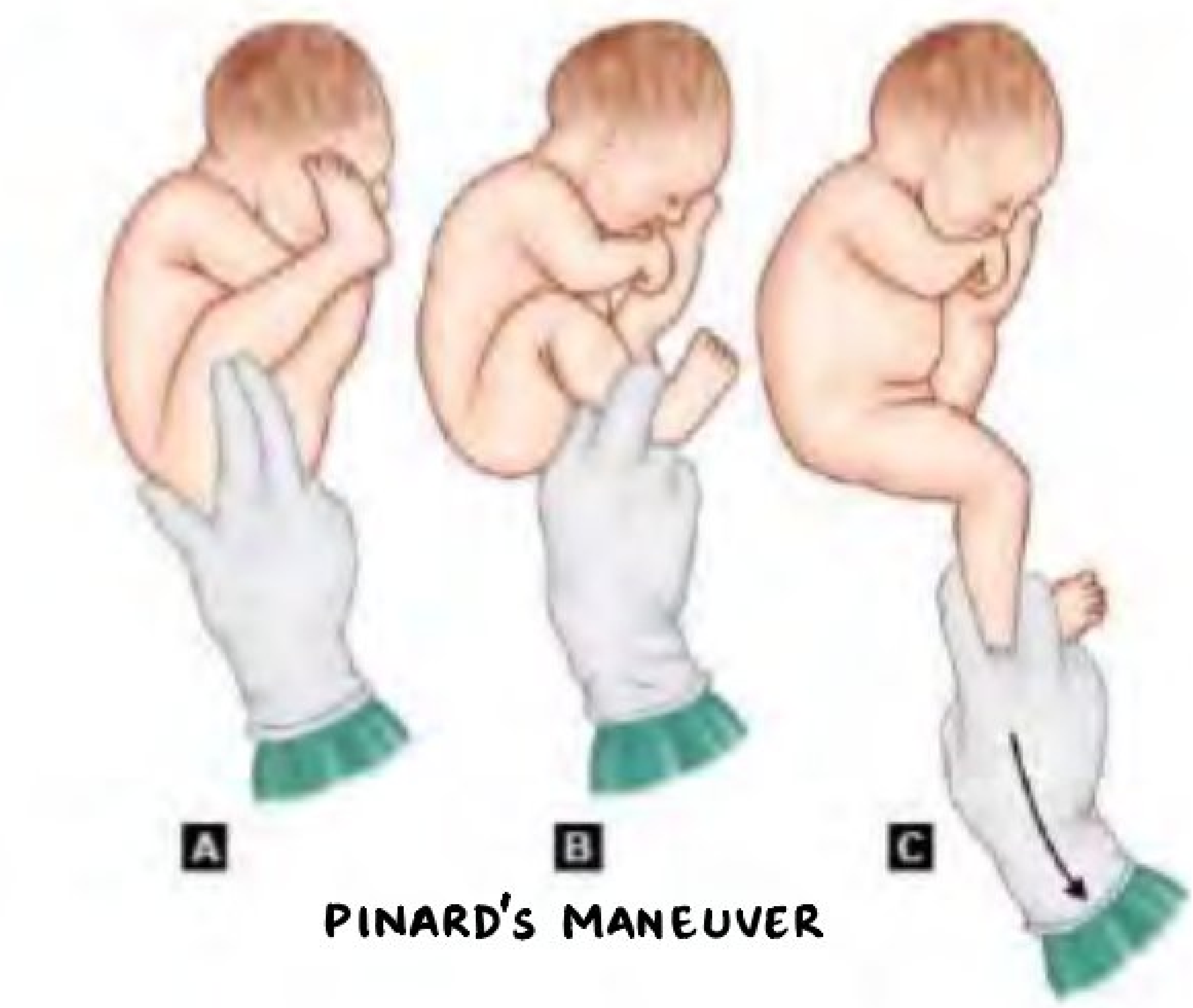
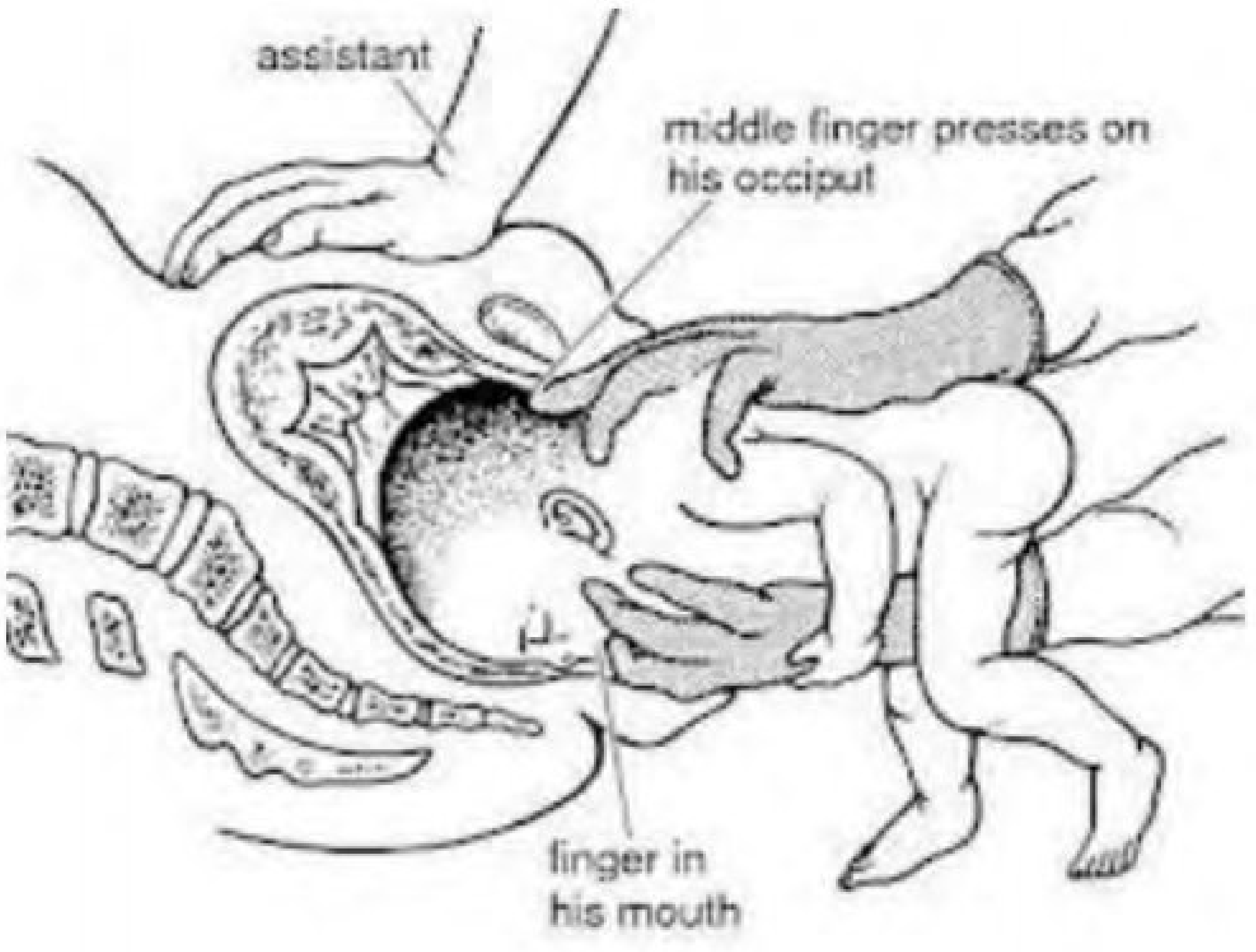


Fig. 5.17 Løveset's manoeuvre


THE BURNS–MARSHALL MANEUVER
a
let him hang until you can see the hair at the back of his neck
b
hold his feet
c
free his mouth and suck him out
d
swing his head clear

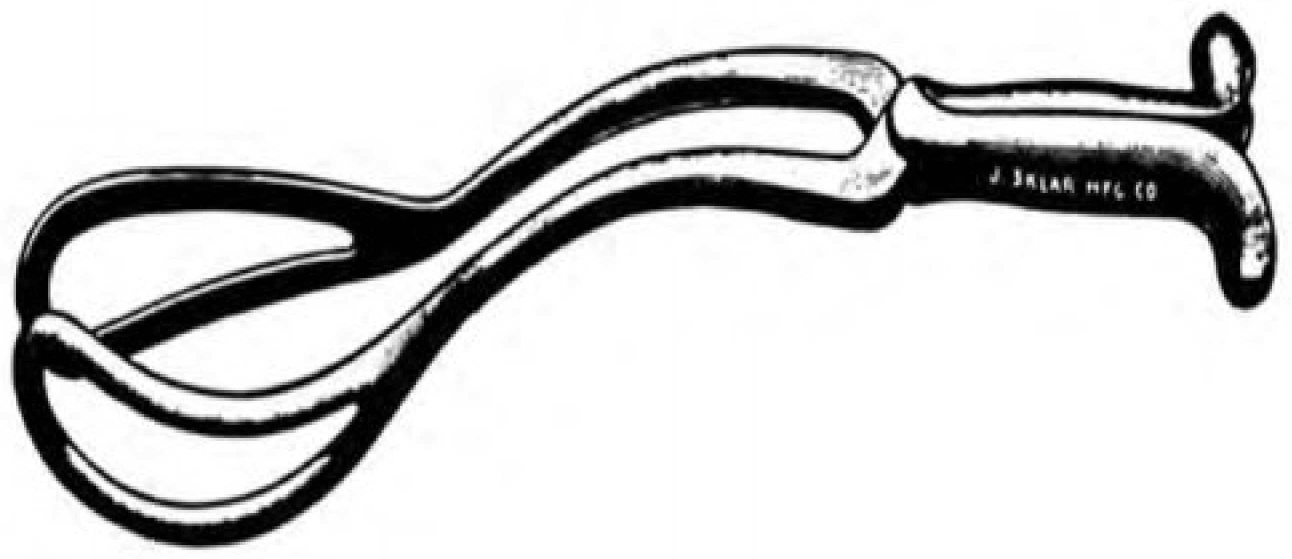


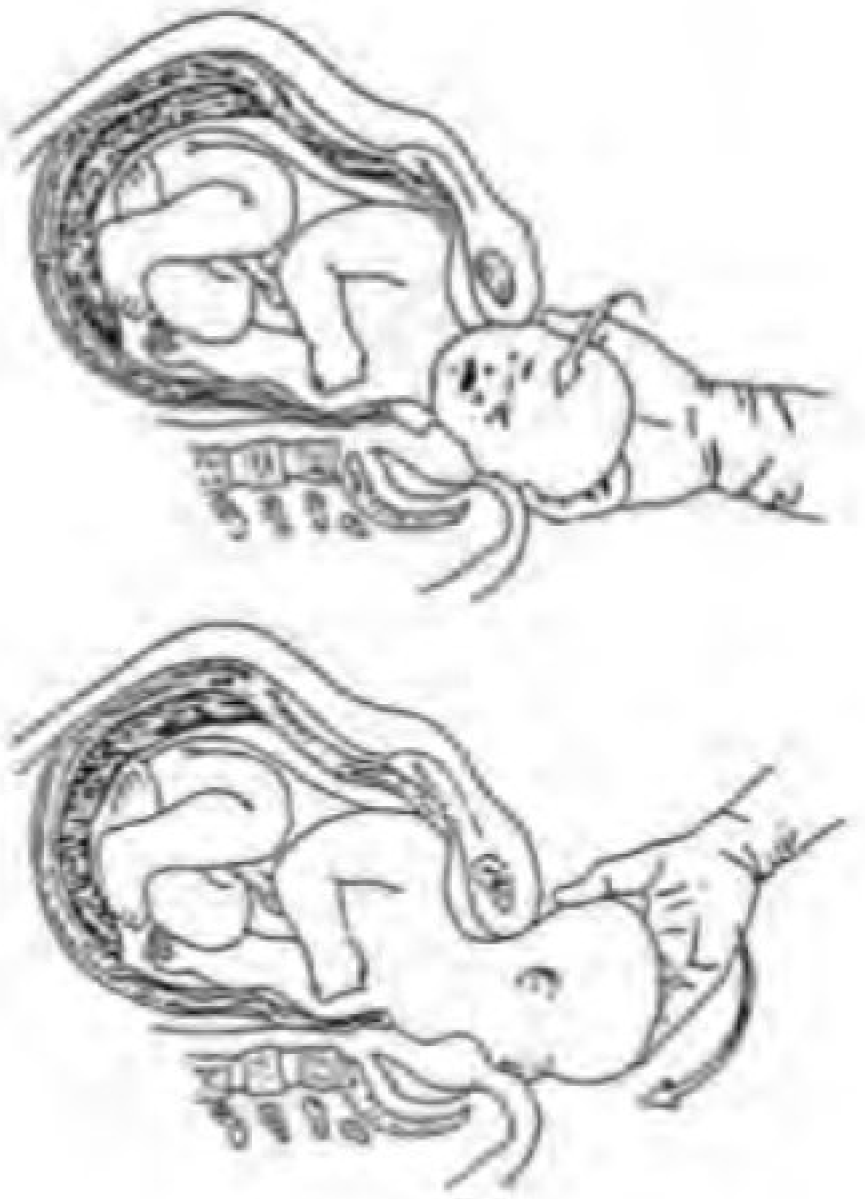
PINARD'S MANEUVER



Mauriceau-Smellie-Veit manouvre

PIPERS FORCEPS





ZAVANELLI MANEUVER

FACE BROW

- S/w Fully Extended Head
- MC cause of Face presentation ⟶ ANENCEPHALY
  ↘ S/w PLATYPELLOID Pelvis
- How to differentiate b/w Face and Breech? (2 Bony prominences + 1 opening)

Face: o ↘ o ↗ o

Breech: o ⟶ o ⟶ o

Risk Factors for FACE / BROW Presentation: Any which prevents FLEXION of Head

1. Multiple tight loops of cord ⟶ Extension
2. Anterior Neck Mass - Eg: CYSTIC HYGROMA

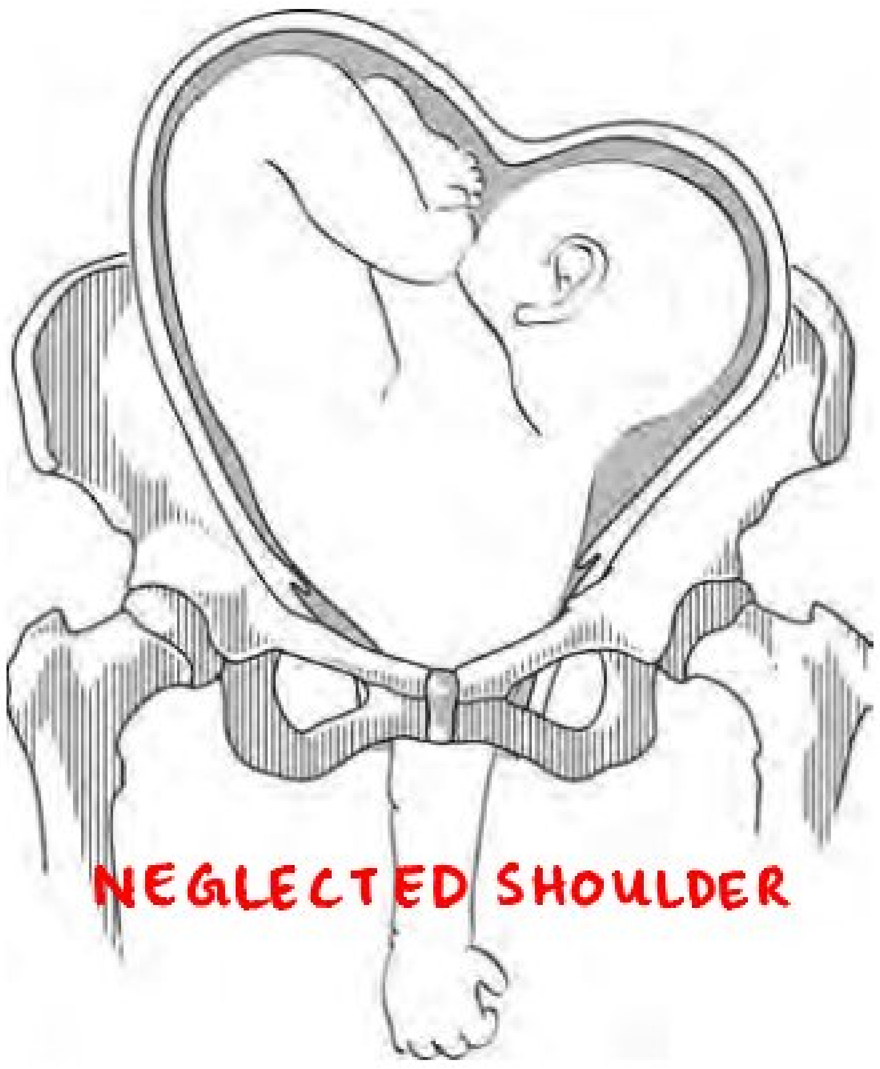
- In Face presentation → MENTO ANTERIOR → Vaginal delivery possible
  - delivery by FLEXION
  - → MENTO POSTERIOR → No Mechanism of V.D
    Mx: by C.S
- If Mento-posterior in early (Latent Phase) Labour
  ↓
  Wait and Watch → Baby will Rotate to Mento-Anterior - delivered by Vaginal delivery

TRANSVERSE LIE

- Shoulder presentation
- a/w PLATYPELLOID Pelvis
- MC cause of PREMATURITY
- MC cause Transverse lie at Term : PLACENTA PRAEVIA
- Transverse lie at TERM → Mx: ECV (Non-Cephalic presentation)
- Transverse lie in LABOUR → V.D is not possible, So Mx. by C.S

NEGLECTED SHOULDER Presentation

- Prolapse of Hand
- FHR Absent } do C.S
- Fetal distress } do C.S
- Older procedure - 1. DECAPITATION of HEAD of Baby
  2. EVISCERATION



CORPORA CONDUPLICATA - double up manner d/t

- Pre-Term
- Old IUD (Maceration)
- Transverse Lie → V.D done

ABNORMAL LABOUR - INSTRUMENTAL DELIVERY

Requirements for Instrumental delivery

F - Fully dilated Cervix

O - No OBSTRUCTION in path

R - Ruptured membranes

C - Good Uterine Contractions

E - Engaged head ; Episiotomy ; Empty Bladder

P - favourable Presentations

CONTRAINDICATIONS

1. CPD
2. Contracted pelvis
3. HIV +ve [NACO]
4. Fetus k/c/o Coagulopathy
5. Fetus k/c/o OSTEOGENESIS IMPERFECTA

- Instrumental delivery is done whenever we want to CUT SHORT 2nd Stage of LABOUR

- INSTRUMENTS used

FORCEPS	VACUUM [VENTOUSE]
Needs training	Much easier to use
Mother is exhausted (Maternal Effort is NOT required)	Maternal effort is required
can be used in	can be used in 1. Heart ds patients
1. Heart ds patients	
2. Fetal distress	2. Fetal distress (in vacuum we will gradually raise pressure
3. Pre-Term	CONTRAINDICATED in
4. After coming head breech	1. Pre-Term
5. Face presentation: MENTO-ANTERIOR but not MENTO-POSTERIOR	2. After coming head breech 3. Face presentation: MENTO-ANTERIOR
Full Dilatation of cervix is MANDATORY	Full dilatation - preferred but not mandatory. can be applied ≥ 6cm
C/I in Non-Rotated Head	can be applied in Non-Rotated Head
More Maternal Injury	more Fetal injury

FORCEPS CLASSIFICATION (Station)

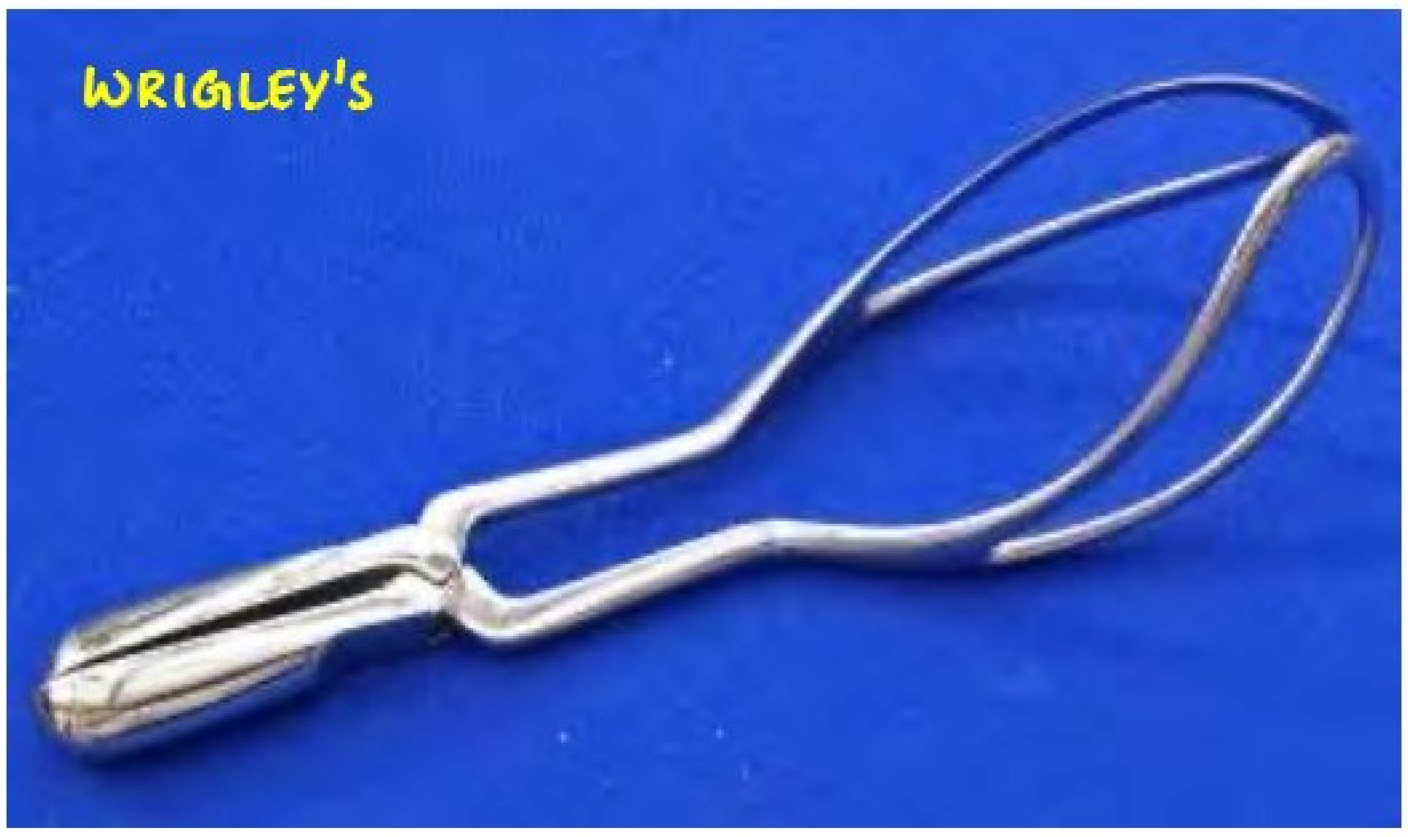
Outlet ⟶ > +3 ⟶ (MC) used 1. WRIGLEY's 2. SIMPSON's 3. TUCKER Mclane

Low ⟶ ≥ +2

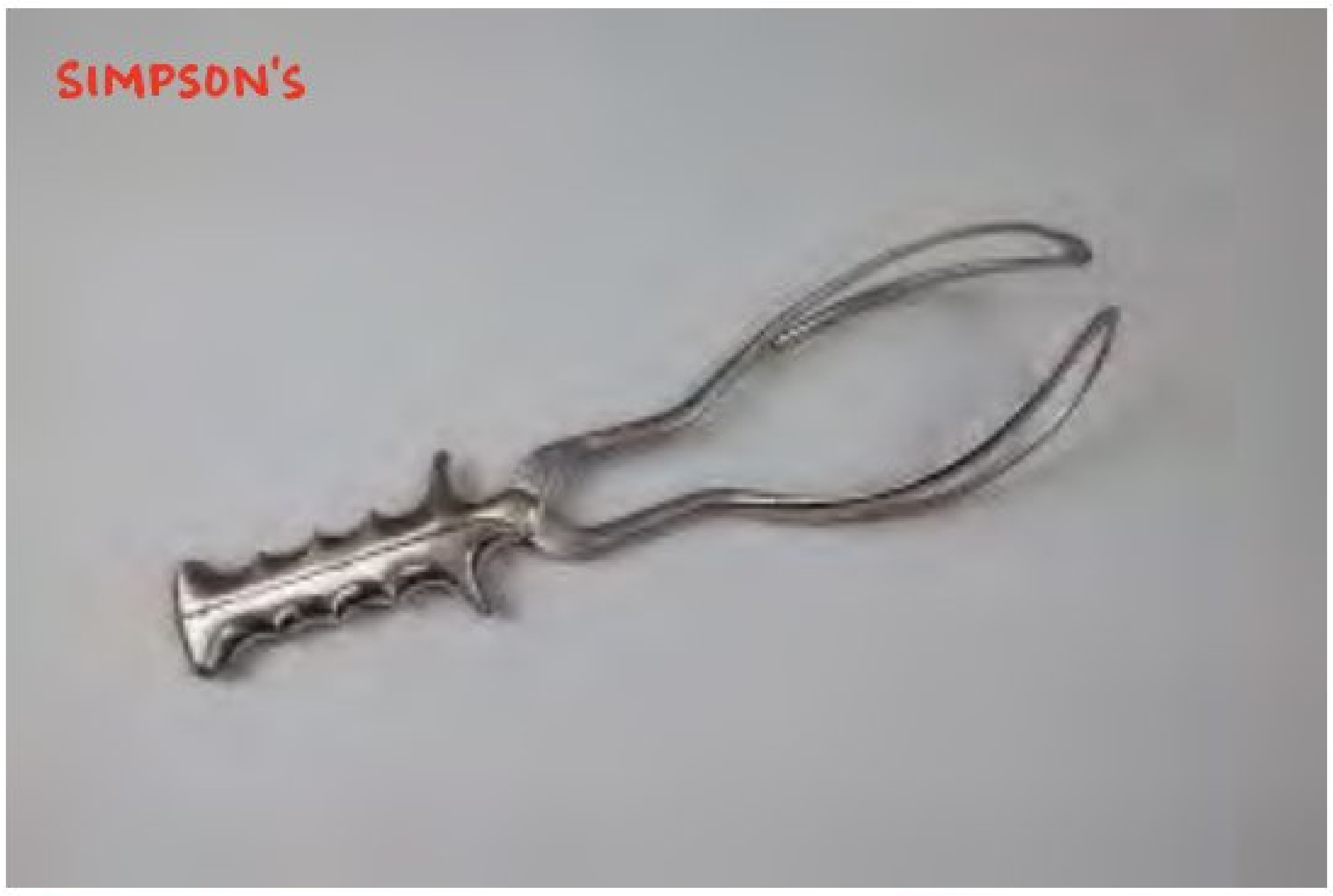
Midcavity ⟶ b/w 0 and +2 } No longer used.

High Forceps ⟶ Above 0

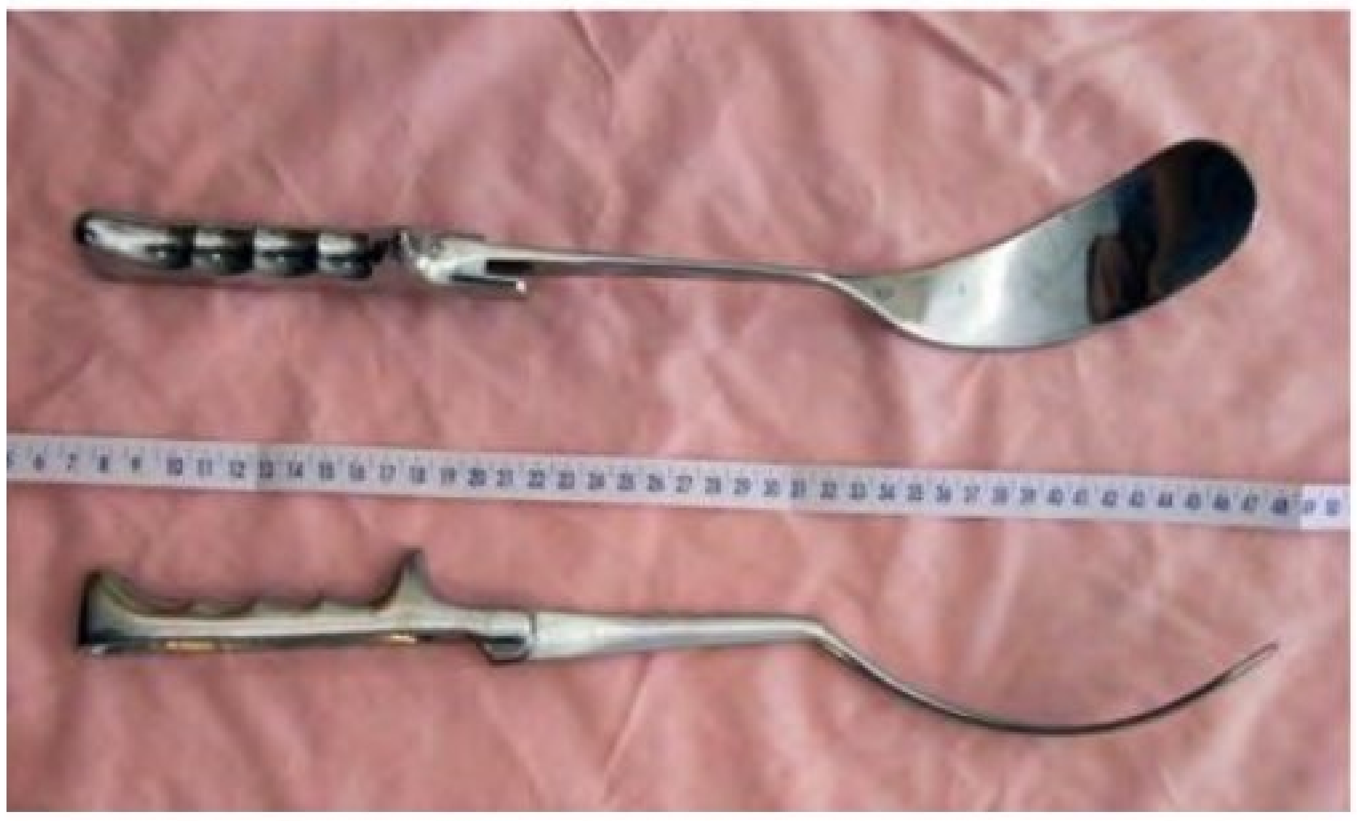
WRIGLEY's - • Short forceps
• English lock < Socket in Ⓛ Hand lower / Ⓡ over



- Inner - Cephalic curve
- upper border - Pelvic curve



Tucker-McLane

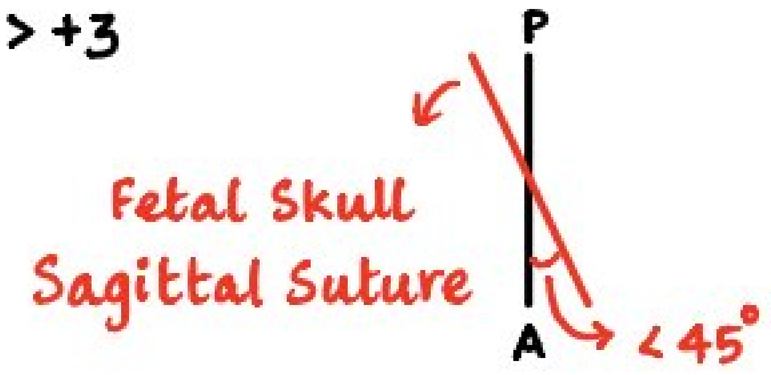


Requirements for OUTLET Forceps

- Scalp should be visible at INTROITUS
- Skull on the pelvic floor
- Head on the perineum

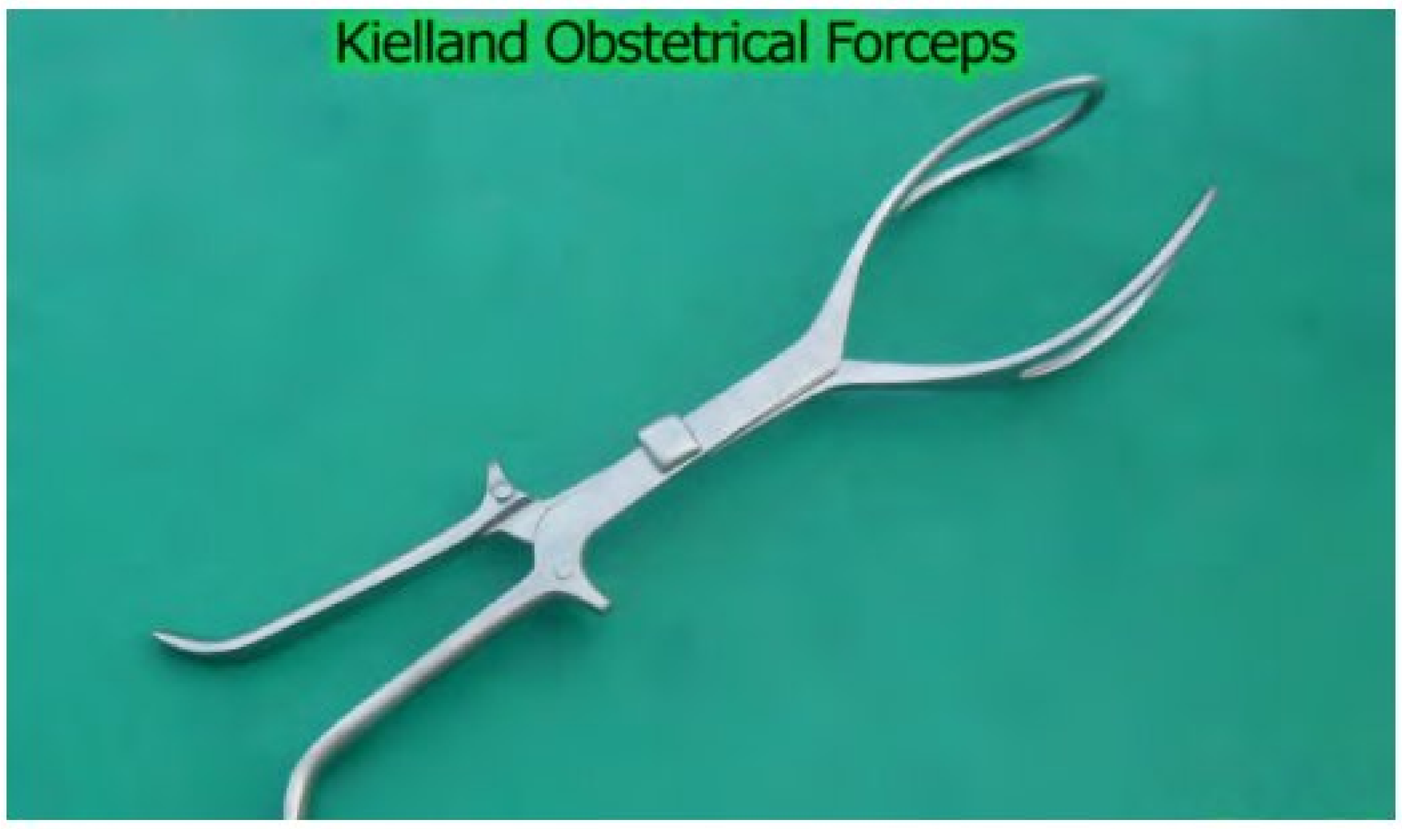
} Station > +3

- Sagittal Suture in AP diameter - Blades should be equidistant from Sagittal Suture
- can be used in case of ROTATIONAL DEFECT < 45°



KEILAND FORCEPS

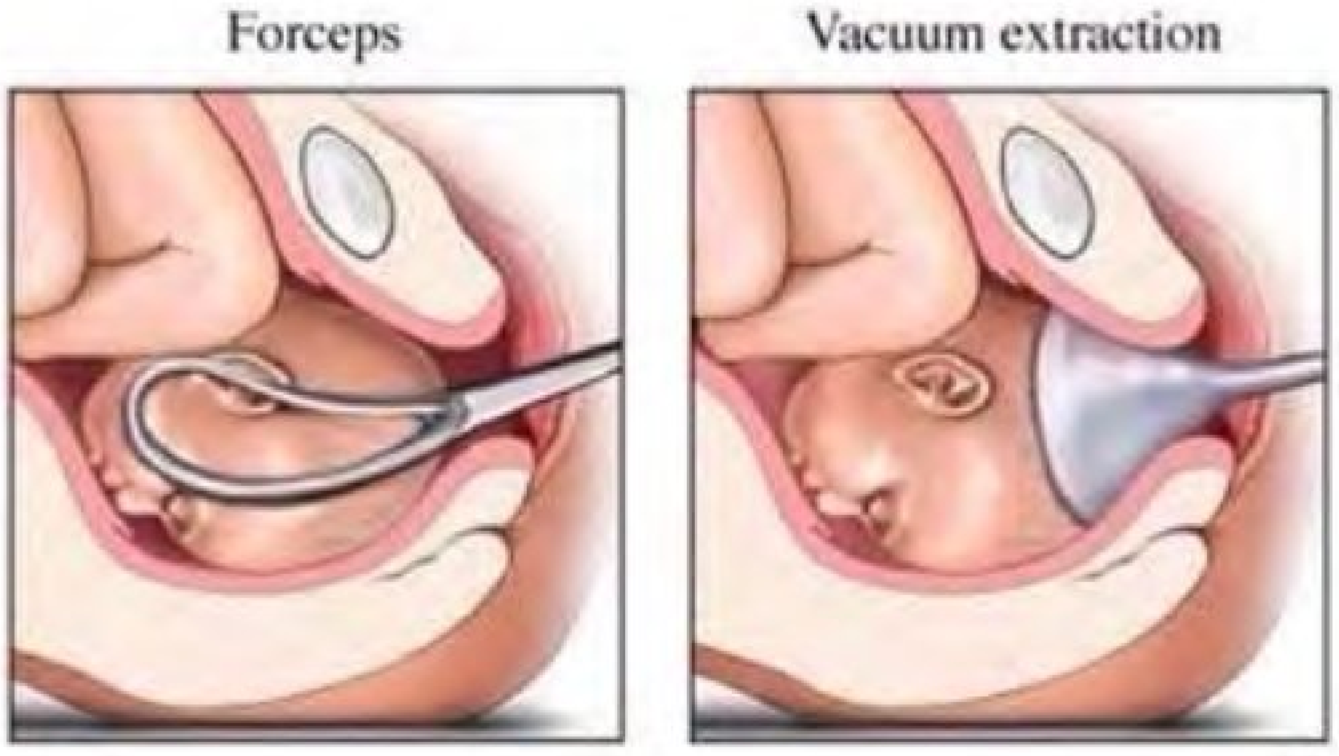
- long
- Sliding lock
- for Rotation



VACUUM

- modern - plastic (SIALISTIC)
- BELL Shaped : (MC) used cup c̲ dia 5-7cm
- Gradual ↑ in Pressure [0.2 → 0.4 → 0.6 → 0.8 Kg/m² or 600 mm Hg]
- Cup applied at a point [FLEXION Point - 3cm Anterior to Posterior Fontanelle or 6cm Posterior to Anterior Fontanelle]
- CORRECT Application → Perimeter of cup touches Post. fontanelle

Instrumental Delivery



- Done only when head of fetus ≥ + 2 Station

- **FORCEPS**

+2 Station
↓
LOW FORCEPS Application

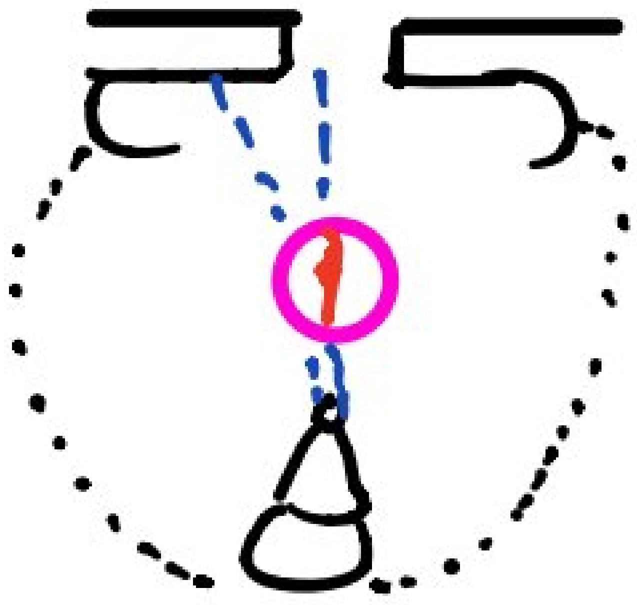
+3, +4, +5 station
↓
Outlet forceps Application
↓
WRIGLEY'S FORCEPS

- When to Apply Outlet forceps? (Pre-Requisites)

- Fetal Scalp visible without separation of mother labia
- Fetal Skull at pelvic floor
- Fetal Head at perineum
- Since Forceps cannot rotate head of the fetus → ∴ Sagittal Suture of fetus should lie in ANTERO-POSTERIOR Direction or Maximum deflection should be < 45°

LOW FORCEPS APPLICATION

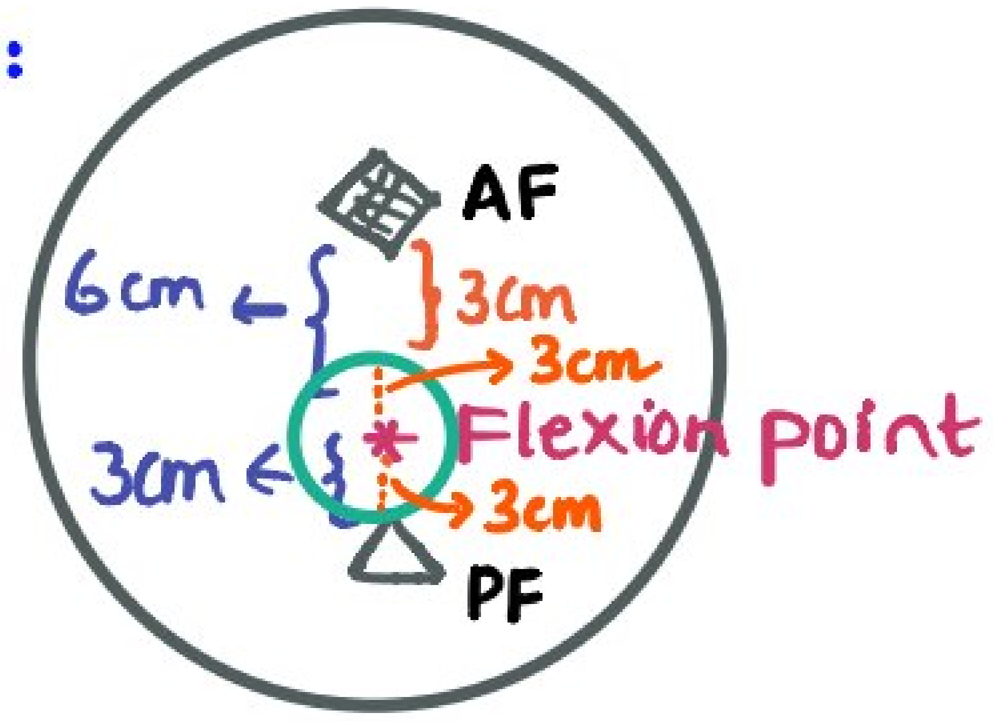
- Direction to pull
  DOWNWARD
  ↓
  FORWARD
  ↓
  UPWARD and FORWARD



OUTLET FORCEPS APPLICATION

- Direction of pull → Forward
  ↓
  upward and Forward

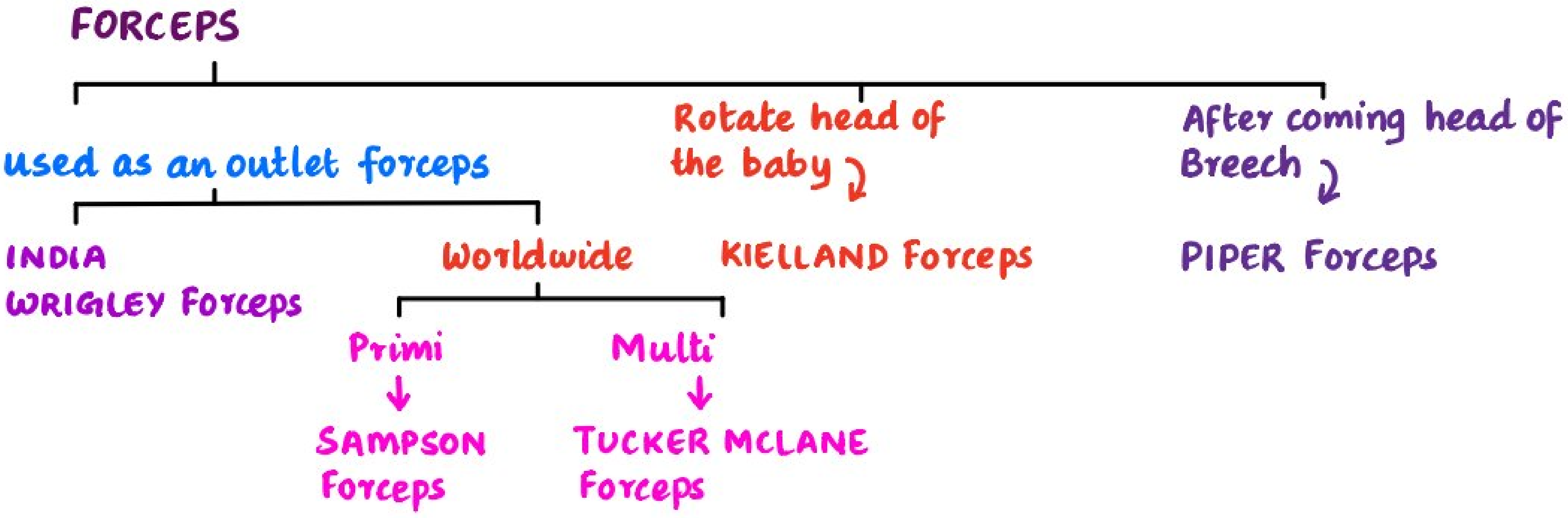
FORCEPS	VACCUM
CRITERIA : F : Favourable position and station O : Os should be fully dilated / Cervix fully dilated	CRITERIA : All the criteria as forceps delivery Except :→ * vaccum is applied when cervix is ≥6cm dilated

R : Membranes should be **R**uptured Head should be **R**otated **C** : Uterus should be **C**ontracting **E** : Head should be **E**ngaged **E**pisiotomy should be given Bladder should be **EMPTY** **P** : Pelvis adequate, No CPD **TRACTION :** PRIMI - 20 Kg MULTI - 13 Kg	* vaccum can rotate head of Baby **PRESSURE :** Initial pressure = 0.2 Kg/cm^2 Maximum = 0.8 Kg/cm^2 **CHIGNON :** It is a iatrogenically created caput succedaneum.
APPLICATION : applied along Occipitomental diameter	**APPLICATION :**  AF 6cm 3cm 3cm Flexion point 3cm 3cm PF * It is applied in such a manner, centre of vaccum cup should lie at FLEXION POINT. Flexion point : It is a point on skull which lies 3cm Anterior to Posterior Fontanelle and 6cm posterior to Anterior Fontanelle - vaccum cup diameter = 6cm - Distance b/w Rim of the cup and Anterior fontanelle = 3cm. Q PGI
Advantages of Forceps : can be used in * Pre-Term deliveries * In Fetal distress * can be used in all those presentation where vaginal delivery is possible → i) vertex ii) Breech → After coming Head of Breech iii) Face → Mento anterior	* Not used in pre-term deliveries * Not used in Fetal distress - can be used only in vertex - Not used in after coming head in Breech - Not used in FACE Presentation
INDICATIONS : 1. Maternal distress 2. Fetal distress	**INDICATIONS :** 1. Maternal distress 2. Prolonged 2nd Stage labour

3. Prolonged 2nd Stage Labour 4. In Heart disease patients / PIH ↓ To cut short the 2nd Stage Labour ↓ K/a Prophylactic use of Forceps	3. In Heart disease patients / PIH In Heart disease pts, Vacuum is preferred over forceps (Because Lithotomy position is not required in vacuum)
COMPLICATIONS : 1. more maternal injury Fetal complications more common c̄ forceps - Facial nerve injury - Brachial plexus injury	COMPLICATIONS : * more fetal injury Fetal complications more common c̄ vacuum - Cephalhematoma - 6th Nerve Injury - Retinal injuries
No. of attempts : Maximum attempts = 3 ↓ Not delivering Failed Forceps ↓ Management → Cesarean section	No. of attempts : Maximum attempts = 3 ↓ Not delivering Failed vacuum ↓ Management → Cesarean section

CONTRAINDICATIONS :

1. Mother is HIV positive
2. Fetus → known case of coagulopathy
3. Osteogenesis imperfecta
4. Contracted pelvis / Cephalopelvic disproportion



FORCEPS

Used as an outlet forceps

INDIA
WRIGLEY Forceps

Worldwide

Primi ↓ SAMPSON Forceps

Multi ↓ TUCKER MCLANE Forceps

Rotate head of the baby ↓ KIELLAND Forceps

After coming head of Breech ↓ PIPER Forceps

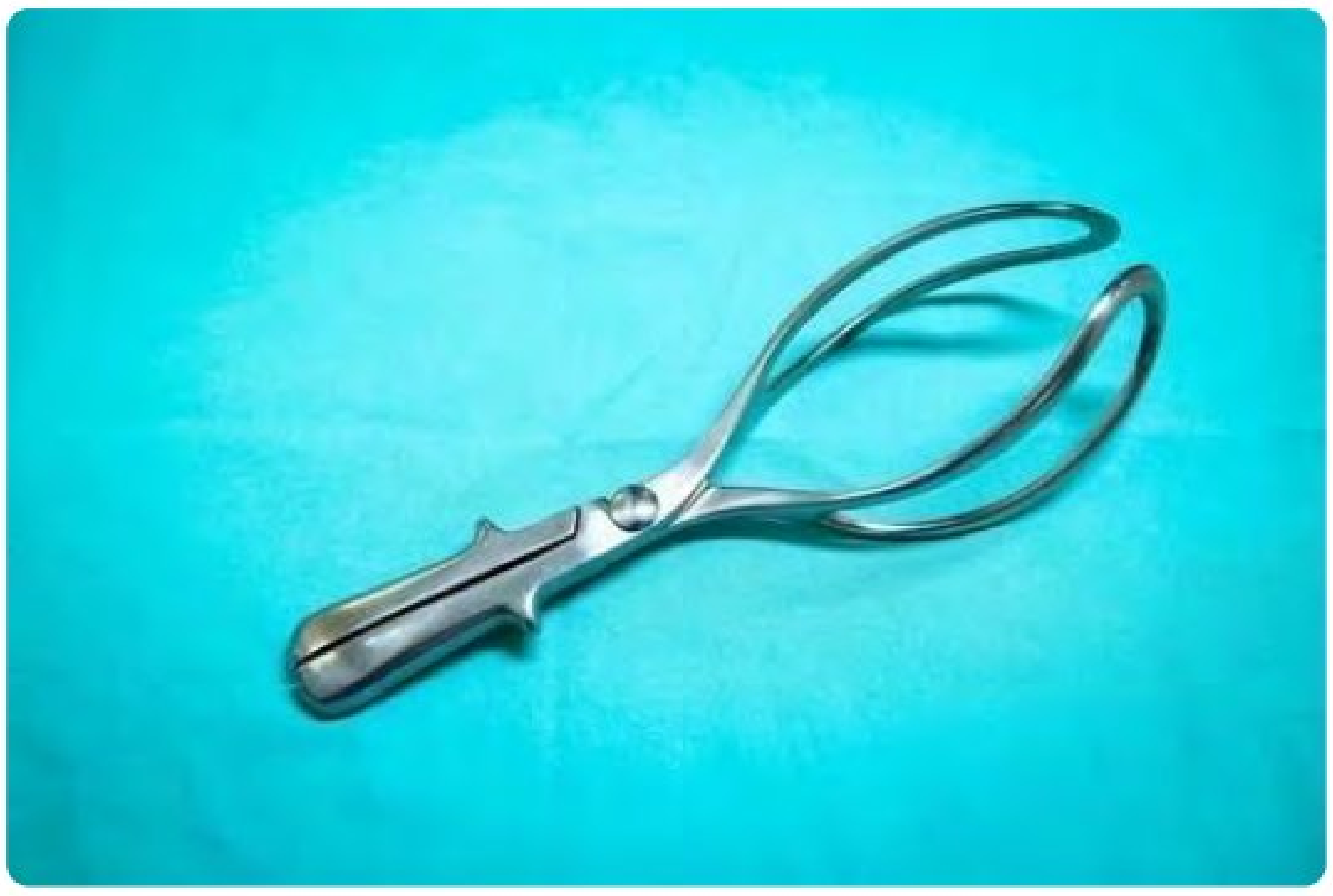
FORCEP Application : Since Fetal position during Labour, (MC) position is

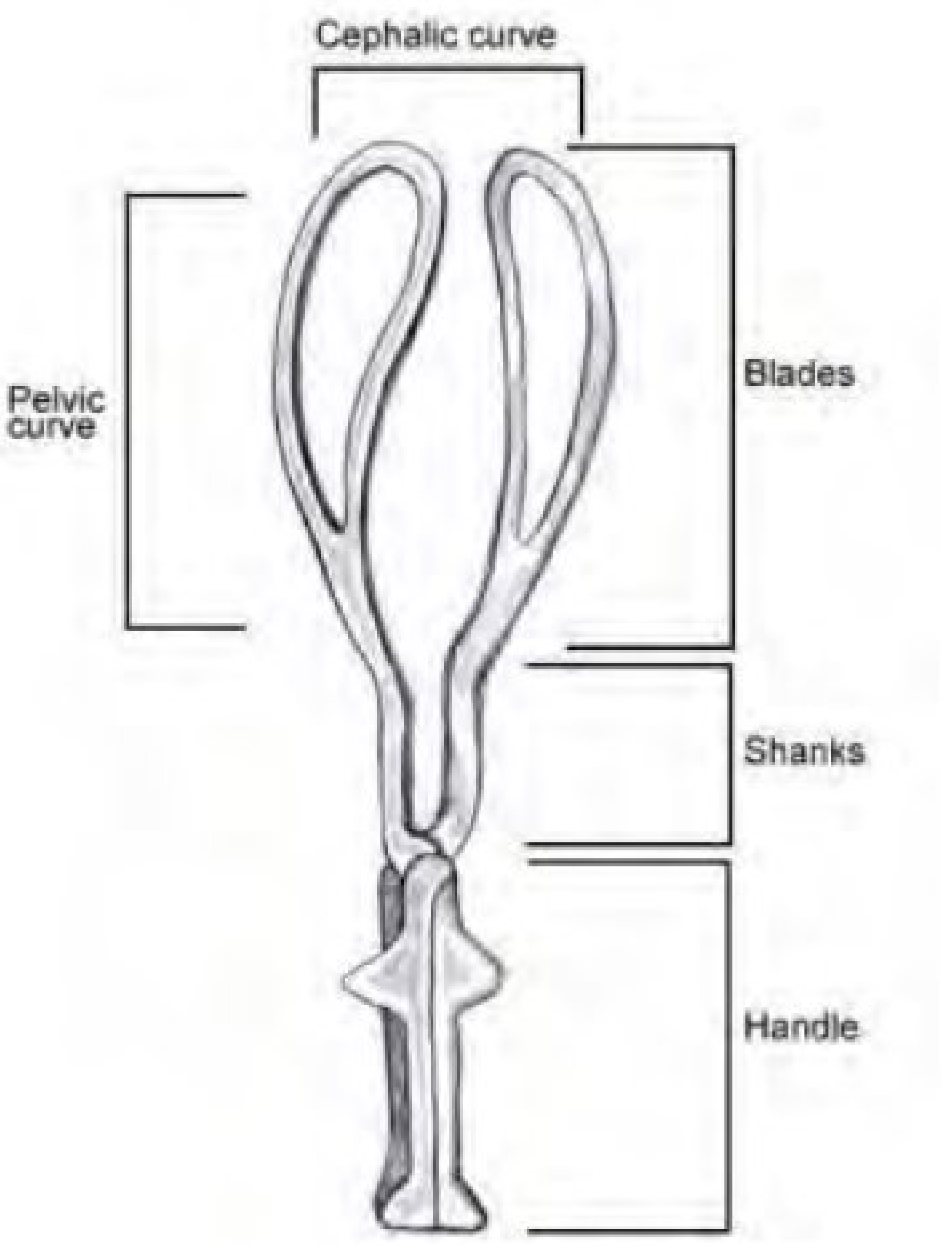
Left Occipito Transverse (LOT) / Left Occipito Anterior (LOA)
↓
1st Left Blade is Introduced.

* But in case of ROT/ROA → Right Blade is Introduced 1st

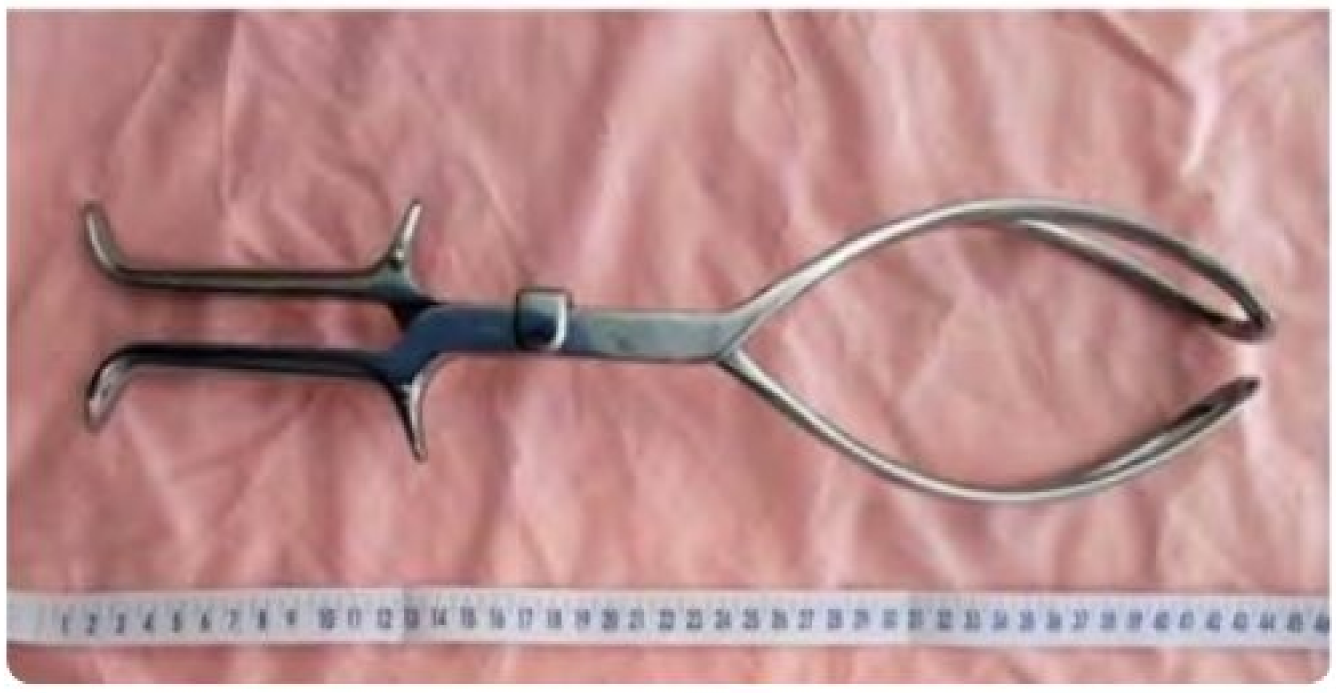
Parts of Forceps:

1. Cephalic curve → Accomodates Fetal Head
2. Pelvic curve → Aligns c̄ Bony pelvis

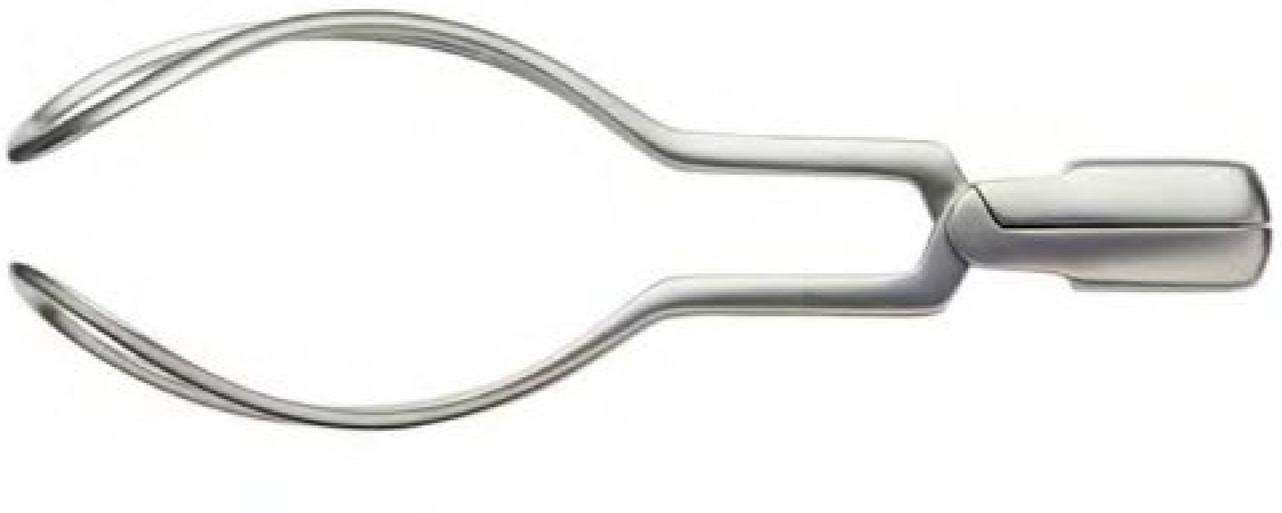




Kielland Forceps

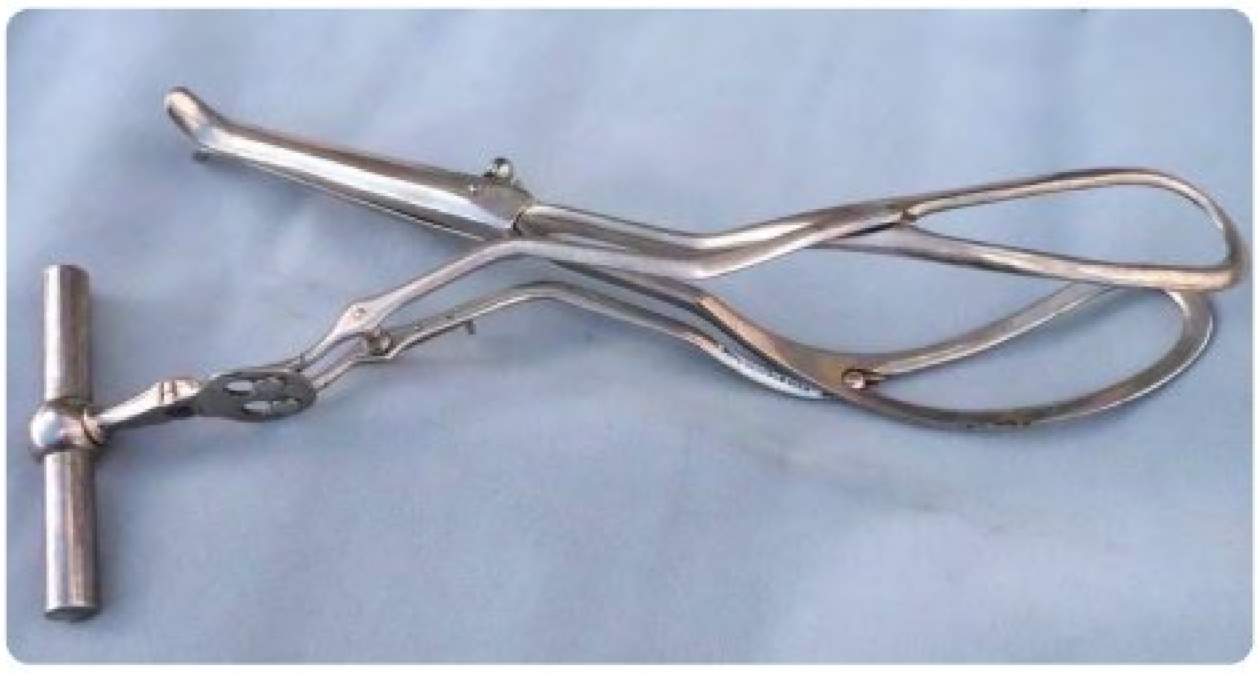


→ Kielland forceps
- Long forceps
- used to deliver a head above +2 station.



Wrigleys forceps

- Outdated forceps
- Has a traction device (to deliver head above + 2 station)



Milne murrays forceps

- Handle - small
- a short, curved forceps

Questions

Q. Traction force required for forceps delivery in Primigravida is

a. 15 Kg   b. 18 to 20 Kg   c. 13 Kg   d. 25 kg

Q. Pre requisite for outlet forceps application all except :

a. Engaged Head b. Fully dilated cervix

c. Uterus contracting d. Station 0 or +1

Q. Forceps delivery is done in all except

a. Mentoposterior b. Deep Transverse Arrest

↓ C. section

↳ Manual Rotation f/b forceps

c. After coming Head d. Maternal Heart disease

↓ Prophylactic use

Q. A forceps rotation of 30° from (LOA) to Occiput anterior (OA) $\underline{c}$ Extraction of the fetus from +2 Station is described as which type of forceps delivery

a. High forceps b. Mid forceps

c. Low forceps d. Outlet forceps

Q. True statement about vacuum extraction of baby (PGI)

a. pressure is maintained b/w 5Kg/cm² to 8 kg/cm²

b. Done when cervix is fully dilated

c. centre of cup should be placed 1cm infront of posterior fontanelle

d. Cup rim should be placed 3cm from anterior fontanelle

Anemia in Pregnancy

IRON METABOLISM IN PREGNANCY

* Total amount of Iron needed in pregnancy → 1000 mg
* Amount of iron needed by fetus → 300 mg
* Amount of iron lost during pregnancy → 250 mg

Daily requirement of iron in pregnancy → 4-6 mg/day

Trimester wise : 1st trimester : 1-2 mg/day
2nd trimester : 4-5 mg/day
3rd trimester : 6 mg/day

* Now since only 10% of dietary iron is absorbed,
↓
So to fulfil requirement of 4-6 mg/day
↓
Amount to be given in diet → 40-60 mg/day
↓
Impossible through diet alone
↓
Hence Iron supplementation is mandatory in pregnancy

ANEMIA MUKHT BHARAT PROGRAMME

Fe = 60 mg + Folic Acid = 500 mcg

Fe ↙ Ferrous Sulphate

60 mg ↓ Prophylactic dose of Iron

↳ It should be started from 4th month of pregnancy.

↳ Continued throughout pregnancy (Ideally) but at least for 180 days during pregnancy & 180 days after delivery
↓
Why? To replenish Iron store

* MC anemia in India is Iron & FA deficiency anemia (Dimorphic Anemia)

ANEMIA IN PREGNANCY

PHYSIOLOGICAL ANEMIA
↓
d/t Hemodilation
↓
{Hb is never < 11gm/dl}

PATHOLOGICAL ANEMIA
↓
d/t other causes like Iron deficiency, FA deficiency etc.
↓
{Hb is < 11gm/dl}

* Anemia in pregnancy is → Hb < 11 gm%
* Severe Anemia in pregnancy is → Hb < 7 gm%
* Very Severe Anemia in pregnancy is Hb < 4 gm%

Trimester wise definition of anemia as per WHO & ACOG

1st Trimester : Hb 11g/dl : Hematocrit <33%
2nd Trimester : Hb <10.5g/dl : Hematocrit 33-32%
3rd Trimester : Hb <10.5 to 11 g/dl : Hematocrit <33%
Post partum : Hb <10g/dl : Hematocrit <30%

IMPORTANT ONE LINERS

i. MCC of Anemia in Pregnancy → Physiological anemia > IDA
ii. MCC of Pathological anemia in preg → IDA (Microcytic Hypochromic)
iii. Ist marker / Most sensitive marker of IDA → S. ferritin
(<30 = IDA
30-40 = Borderline)
iv. MCC of Megaloblastic anemia in pregnancy → FA deficiency anemia
v. Dose of FA in pregnant female c̄ megaloblastic anemia → 1g /day
vi. MC Anemia in pregnant female after Beritric Surgery → Vit B12 def.
vii. MC Indirect cause of maternal mortality in India → Anemia
viii. MC Type of anemia in pregnancy in under → Fe & FA deficiency Anemia (Dimorphic)

Screening For Anemia in Pregnancy

1. Should be done in all pregnant females

2. At 1st antenatal visit & Repeated b/w 24-48 wks
3. By doing complete blood count

TREATMENT OF IDA

1. Oral Iron

Therapeutic dose :- 2-3 tablets of Iron /day
↓
Give till blood parameters become normal

* 1st blood parameters ↑ after giving oral Iron → Reticulocyte count start ↑ after 7 days, Maximum after 10 days
* Hb Increases after 3 wks
* Rate of ↑ of Hb : 0.7 to 1gm/wk

⇓
Then put patient on maintainance dose
i.e 60mg (1 tab)/day throughout preg + 180 days after delivery

Problems of Oral Iron

i. Poor oral compliance
ii. GI S/E
iii. It can't be used in pt c̄ inflammatory Bowel diseases like Ulcerative collitis, Crohns disease
iv. Can't be used after bariatric surgery

2. Parenteral Iron (i/v)

i. Low molecular wt dextran
ii. Iron Sucrose
iii. Ferric carboxymaltrose.

Remember → Rate of ↑ of Hb after parenteral iron is same as oral iron

Advantage →
i. Not patient dependent
ii. Large amount can be given in a single infusion (100 mg IV)
iii. No GI S/E
iv. can be used in pts of IBD & after Bariatric Surgery

CALCULATION OF DOSE OF PARENTERAL IRON

Assume :
- Blood volume = 65 ml/kg
- Hb to be corrected uptill 14 gm
- Each gm of Hb : 3.3 mg of Iron

Dose of Fe (mg) = Body weight x (14 − Hb of pt) x 2·145 + Iron to replenish store

Volume of drug to be given = Above value ÷ C
where C is concentration of Iron/ml in that product

Case ① Suppose : 60kg pregnant female c̄ Hb 8
No need of iron stores
Using iron sucrose = 20mg Iron/ml

⇒ Hb deficit $= 60 \times (14 - 8)\, 2.145 = 772$ mg of Iron

Dose of iron inj needed (ml) $= \frac{772}{20} = 38.6$ ml

TO REPLENISH FE STORES MOST OF TIME WE USE 250mg Fe

Case ② Suppose : Pregnant female c̄ Hb 6 at 36 wks of pregnancy
⇓
WHAT IS NEXT STEP ?? ⇒ Blood transfusion

Indications for Blood transfusion in Pregnancy

i. Pregnancy < 34 wks + Hb < 5gm%
ii. Pregnancy ≥ 34 wks + Hb < 7gm%
iii. at Labor Hb < 7 gm%
iv. Acute Hemorrhage in pregnancy causing Hb < 6gm%
v. Any time if pt has heart failure c̄ low Hb levels

HEART DISEASE IN PREGNANCY

Pathological 1. Orthopnea / Paroxysmal Nocturnal Dyspnea

2. Chest pain

3. Progressive breathlessness (EXCERTIONAL)

SIGNS

1. Systolic murmur (>Grade 2)
2. Cyanosis / Clubbing
3. CHF / Rhythm Ab Ⓝ
4. Loud P_2
5. Wide Split S_2
6. Cardiomegaly

INVESTIGATION - ECHO

- MC Heart ds : MITRAL STENOSIS
- MC Congenital Heart ds in Pregnant : ASD
- MC Congenital Valvular Heart ds : MVP

HIGH MORTALITY (Poor Prognosis)

↳ 50% seen with

1. Marfan's Syndrome c̄ involvement of Aortic Root
2. Coarctation of Aorta c̄ AORTIC Valve
3. EISSENMENGER's Syndrome
4. Severe MS (valve area < 1.5 cm^2)

   Severe AS (valve area < 1 cm^2)
5. NYHA Grade 3/4
6. Ejection Fraction < 30%

} TERMINATION OF PREGNANCY

CONGENITAL LESIONS c̄ GOOD OUTCOME

1. ASD
2. VSD
3. Ebstein anomaly
4. Corrected TOF
5. MVP

In case of Severe MS → In Pregnancy : Valve Replacement Surgery - not done because 30% mortality

- We should do BALLOON VALVOTOMY ≥ 20wks

↓

Ideal

↓

They should undergo Valve Replacement Surgery prior conception

↓

1. METALLIC - Lifelong Anticoagulant needed
2. BIOPROSTHETIC - Replaced every 10yrs

↓

1-12wks : On HEPARIN - because Warfarin is TERATOGENIC : Warfarin embryopathy CHONDRODISPLACIA PUNCTATA

12-36wks: On WARFARIN - more potent and because patient is HIGH RISK

>36wks upto onset of LABOUR: On HEPARIN don't want to give Warfarin close to labour
If given can cause bleeding in baby.

Q. K/c/o Heart ds, Preg. woman on Warfarin Rx goes into Pre-Term Labour. What next?

→ should be delivered C·S

Q. Preg. women c̄ h/o baby c̄ Warfarin embryopathy. What next?

Ans: Avoid Warfarin in 1st TM

RULES

1. Women c̄ Heart ds : VD is preferred

2. CS is reserved for Obstetric Indications

3. Heart ds in which C·S is preferred

   a. Marfan's Aortic Root
   b. Coarctation c̄ Aortic valve
   c. Severe AS/Severe AR
   d. AORTIC DISSECTION
   e. Woman on Warfarin goes in Labour
   f. Refractory CHF

4. If women c̄ heart ds taken up for C·S.
   Anesthesia given: EPIDURAL > NEURAXIAL

   exceptions above mentioned ⓐ → ⓕ in these conditions we give G.A

5. In V·D - Spontaneous >> Induction.
   IOL is NOT C/I

6. Preferred Analgesia even in Heart ds: EPIDURAL

7. Restrict i.v fluids at 75ml/hr

8. Restrict P/v exam

9. Membranes Rupture → Prophylactic Ab for I.E
   (Ampicillin + Gentamicin)

   AHA: V.D is not an Indication for Prophylaxis of I.E

10. Cut short the 2nd stage : Forceps >> Vacuum

11. Risk of CHF Highest - Immediate Post Partum
    ↳
    as soon as woman delivers give i.v FUROSEMIDE

C/I in Heart ds → METHERGINE

Diabetes in Pregnancy

CHANGES IN CARBOHYDRATE METABOLISM DURING PREGNANCY

Insulin Resistance During Pregnancy

↓ d/t

- Mainly
  HPL
- Other Hormones
  - Estrogen
  - Progesterone
  - Cortisol
- MC
  ↓
  at 24-28 wks of pregnancy

* Fetus dependant on mother for glucose requirement

MOTHER

Glucose ↓ GLUT1 & GLUT3

FACILITATED DIFFUSION

↓

FETUS

* In pregnancy there is
  - Fasting Hypoglycemia
  - Post prandial - Hyperglycemia
    (d/t insulin resistance)

* Glycosuria is normal during pregnancy
* Maternal insulin can not cross placenta ∴ Insulin DOC for treating Diabetes during Pregnancy
* Fetus start producing Insulin at 12 wks of Pregnancy

so if Maternal glycemia

↓

Fetal Hyperglycemia

↓

β cells of Pancreas of fetus → Hyperatrophy

↓

1. ↑ Growth of Fetus
   ↓
   Macrosomia
2. ↓ Surfactant production
   ↓
   ↑ ROS
3. After Birth
   ↓
   Neonatal Hypoglycemia

HORMONE Responsible for growth of fetus : INSULIN

DIABETES IN PREGNANCY

Diabetic female has conceived
⇓
OVERT DIABETS

Female was normoglycemic before pregnancy
↓
During pregnancy d/t Insulin Resistance
↓
Becomes Diabetic
↓
Gestational Diabetes

*blood sugar levels ↑ from day 1 of pregnancy

*Blood sugars levels of ↑ after 24-28 wks of pregnancy

In diabetes ⇒ ↑ blood sugar
⇓
↑ Free radicals
⇓
Congenital Malformation

* Free radicals are formed from Day 1
⇓
↑ chance of congenital malformation

* Free radicals will be formed after 24-28 wks of pregnancy
⇓
By this time organogenesis is complete
⇓
No congenital Malformation seen in babies of gestational Diabetes

OVERT DIABETES

- FBS ≥ 126 mg/dl
- RBS ≥ 200 mg/dl
- HbA1C ≥ 6.5%

* Test which predicts the chances of congenital malformation in babies of overt diabetes mother
⇓
HbA1C

- <6.5% → No Chance
- 6.5-9.5% → 5%
- ≥9.5% → 20%

* Best test which detects congenital malformation in babies of Diabetic mother ⟶
⇓
TIFFA/ Level 2 USG at 16-20 wks of pregnancy.

TIFFA should be done in gestational diabetic and overt diabetic pregnant female

METHODS TO PREVENT CONGENITAL MALFORMATIONS

1. By strict control over glucose
2. FA supplementation ⇒ 400mcg/day

DOC for treating overt diabetes
↓
INSULIN

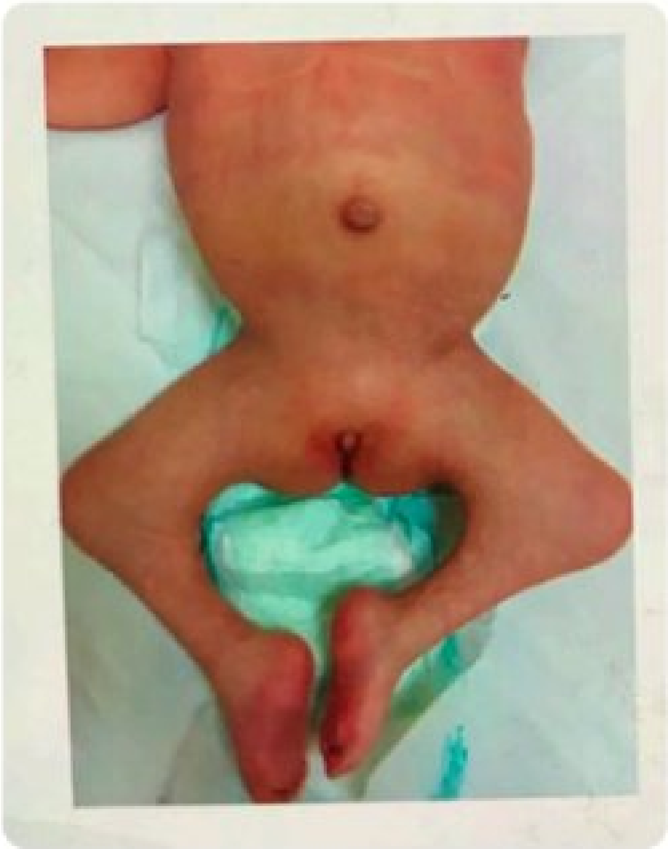
All oral Hypoglycemic drugs are contraindicated

- MC System involved in congenital malformation : CVS > CNS
- MC Congenital anomaly seen : VSD > NTD
- Most specific anomaly seen : Caudal Regression Syndrome / Sacral Agenesis (Rare)
- Most specific cardiovascular anomaly → TGA
- MC cardiac anomaly seen in babies of diabetic mother : VSD
- MC Cardiovascular finding seen in babies of diabetic mother : HOCM
- Least common cardiovascular anomalies seen in babies of diabetic mother : TOF

DIAGNOSIS OF GESTATIONAL AGE

2step approach
by ACOG

1step approach
- Recommended by WHO (ADA & IADPSG)
- Recommended by DIPSI


Caudal Regression Syndrome

TWO STEP APPROACH

-Recommended by ACOG

1st STEP: Screening Test : Glucose Challenge test (GCT)

Time = 24-48 hrs
No fasting Required
Method : 50 gms glucose to patient
↓ 1hr
Check Sugar Level

<140 mg/dl → Nothing
>140 mg/dl → Do Next step, i.e Diagnostic step
≥180 mg/dl → Diabetes confirmed

NOTE:

-ACOG (2017) Recommends universal screening in all pregnant female irrespective of Risk factors.

-In low risk female → GCT
to be done only once b/w 24-48

-In High Risk female → GCT
to be done at 1st antenatal visit & repeated at 24-28 wks

High Risk Females

- Obese female
- Age > 30 yrs
- Previous h/o GDM
- Family h/o Diabetes
- Previous h/o congenital malformation in baby

- Previous h/o still birth in baby
- Previous h/o of IUD in baby
- Previous h/o of ≥ 3 Abortions
- Previous h/o of macrosomia
- If present pregnancy she has any complication related to diabetes
  Eg: Polyhydramnios

2nd STEP: Perform a 3hr 100gm OGTT (Glucose Tolerance Test)

- Preparation: over night fasting

↓ (8-10 hrs)

1st sample = FBS Sample
↓
Give her 100gm of Glucose
↓
2nd Sample : 1 hr PP
3rd Sample : 2 hr PP
4th Sample : 3hr PP

} Total 4 Samples

If any 2 or more than 2 values are abnormal → Confirmed Diabetes

How to decide whether value is abnormal or not ?
↓
Criteria to be followed

1. Carpenter & Couston Criteria

Fasting → 95 mg/dl
1hr → 180 mg/dl
2hr → 155 mg/dl
3hr → 140 mg/dl

2. National Diabetes Data group Criteria

+10 For fasting, 1hr, 2hr
+5 For 3 hrs.

Upper limit of normal values in GTT: 3hr 100gm GTT (AOCG)

	Carpenter & Couston Criteria	National Diabetes data group Criteria
Fasting	95 mg/dl	105 mg/dl
1hr PP	180 mg/dl	190 mg/dl
2hr PP	155 mg/dl	165 mg/dl
3hr PP	140 mg/dl	145 mg/dl

2. **One step approach** – Recommended by WHO

No need of screening test

↓

Directly at 24-48 wks ⇒ ask the patient to do vernight fasting (8-10 hrs)

↓

1st Sample = FBS Sample

↓

Do 2hr 75gm OGTT

↓

Give 75 gm oral glucose

↓

After 1hr ⟶ 2nd Sample

After 2hr ⟶ 3rd sample

Total Samples = 3 (if one or more than one value is abnormal pt is said to be diabetic)

Criteria for 75gms of GTT

Fasting ⟶ 92

1hr ⟶ 180

2hr ⟶ 153

Note : Unlike AOCG, who recommends test only in females with any Risk factor

NOWER DAYS : DIPSI CRITERIA (following in India)

- No fasting needed
- Whenever female comes for antenatal visit (Not necessory 24- 28 wks)

↓

Irrespective of previous meal

↓

Give 75gm of Glucose

↓

After 2hrs blood sugar level

>120 → Glucose intolerance

≥140 → Gestational DM

≥200 → Arrest Diabetes

- This test has to be done in all pregnant female

MANAGEMENT OF DIABETES

1st step : Diet modification + Exercise

↓

For 2 wks
↓
Aim for metabolic goals

Fasting : 70-95
1hr PP : <140
2hr PP : <120
HbA1C : <6.5

↓
Not attained by 2wks
↓
Start INSULIN (DOC)

- * DOC for treating Diabetes in pregnancy - INSULIN
- * During pregnancy Insulin dose is increased
- * During Labor (Patient → Liquid diet /Nil oral) → Dose of insulin ↓
- * Oral hypoglycemics are C/I during pregnancy
  - i) Because they can cross placenta
  - ii) Because during pregnancy = strong drug is INSULIN (Diabetogenic state)

2 oral hypoglycemics which are not C/I during pregnancy
Can be used in Gestational Diabetes
1. GLYBURIDE
2. METFORMIN → Not teratogenic
→ Dangerous S/E
↓
Lactic acidosis

Fetal survellance : Begins by 32-34 wks. If growth restriction is also seen → 28 wks

- Overt Diabetes + GDM Patient on insulin
↓
Admit at 34wks

- Methods of survellance :
  - * Daily Fetal Kick count
  - * Biweekly NST
  - * Weekly BPS

Time of Delivery

AIIMS Nov 18 Q
- GDM controlled on Diet :→ ≥39wks upto 40wks + 6days
- GDM uncontrolled by Insulin → ≥39wks to 39wks + 6 days
- Overt diabetes → 37 wks to 37wks + 6 day
- Mode of delivery → Vaginal Delivery
  - If wt of fetus is ≥4.5 kg in diabetic female
    ↓
    Then go for cesarean section

COMPLICATIONS OF DIABETES DURING PREGNANCY

MATERNAL COMPLICATIONS

- ↑ Infections
  ↓
  Asymptomatic Bacteremia, UTI
  ↓
  - ↑ Preterm Labor
  - PROM ↑
- Big Placenta
  - Placenta Previa
  - ↑ PIH
- Hyperglycemia of fetus
  ↓
  POLYURIA
  ↓
  Polyhydramnios
  ↓
  PROM
  Preterm Labor
  Malpresentation
  Abruptio placenta
  PPH
- In Future
  ↓
  ↑ chances of Diabetes
  ↓
  all patient after delivery b/w 4-12 wks of delivery do GTT

FETAL COMPLICATIONS

1. Fetal Hyperglycemia ⇒ ↑ insulin
2. Macrosomia

↑ in weight of fetus ≥4.5 kg
In India ≥4kg

Risk Factor :
- Male Baby
- Post dated pregnancy
- Diabetes in mother

Which USG Parameter can detect macrosomia →
Abdominal circumference → ≥35cm → Macrosmia

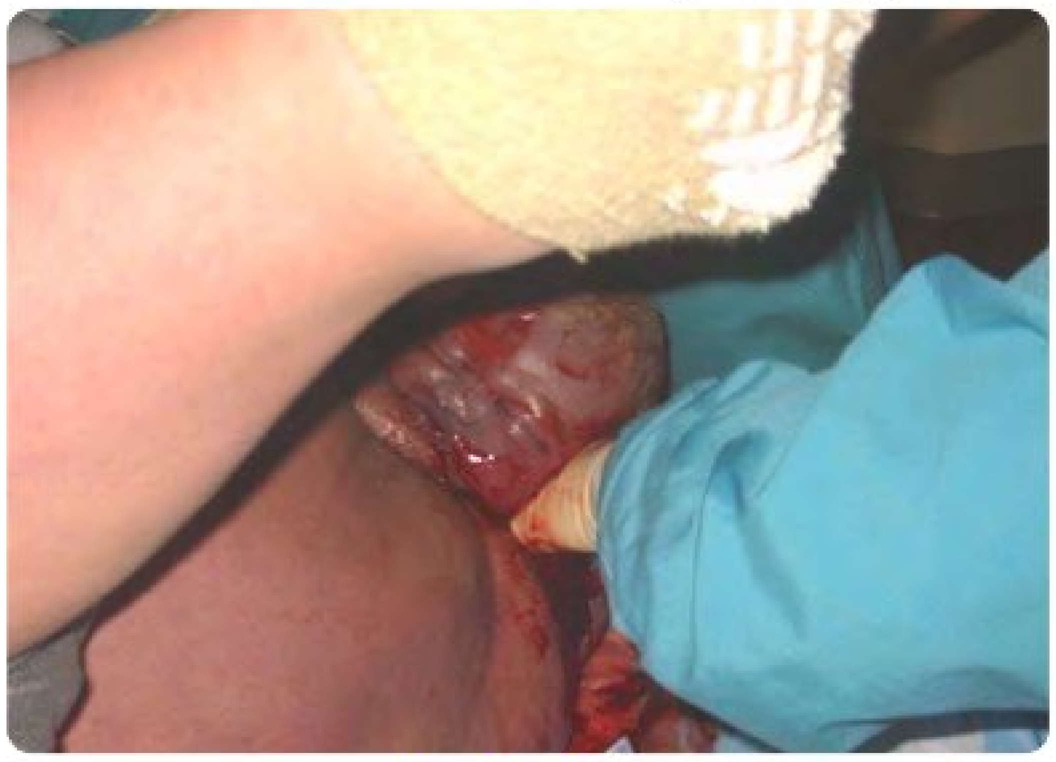
*Organ least affected by macrosomia : Brain
*Management : Cesarean section is done

- wt of fetus ≥45kg in diabetic female
- Wt of fetus ≥5kg in non diabetic female

*During vaginal delivery macrosomia can lead to → Shoulder Dystocia

3. Shoulder Dystocia

- Delay in delivery of shoulder >1 min after delivery of head
- Sudden pulling back of head towards perineum ↓ k/a TURTLE SIGN



Turtle sign

-Risk Factors for shoulder dystocia

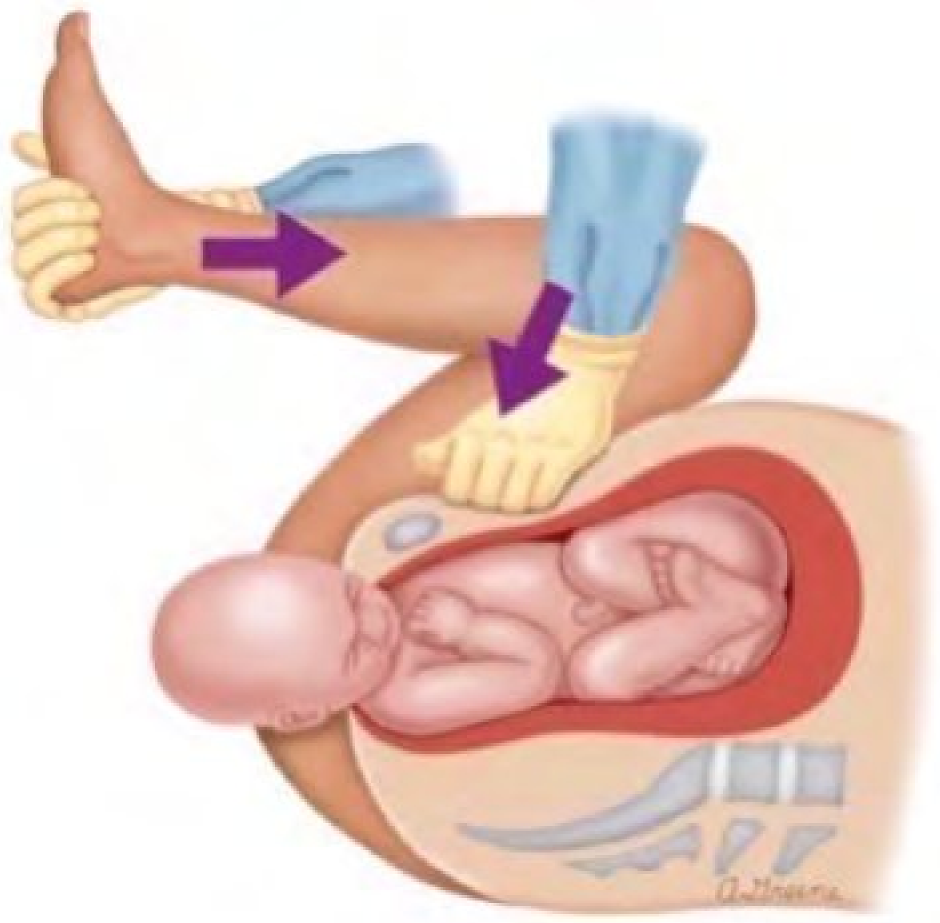
D = Maternal Diabetes
O = Maternal Obesity
Fetal obesity - Macrosomia
P = Post dated Pregnancy
A = Anencephaly (Pseudo shoulder Dystocia)

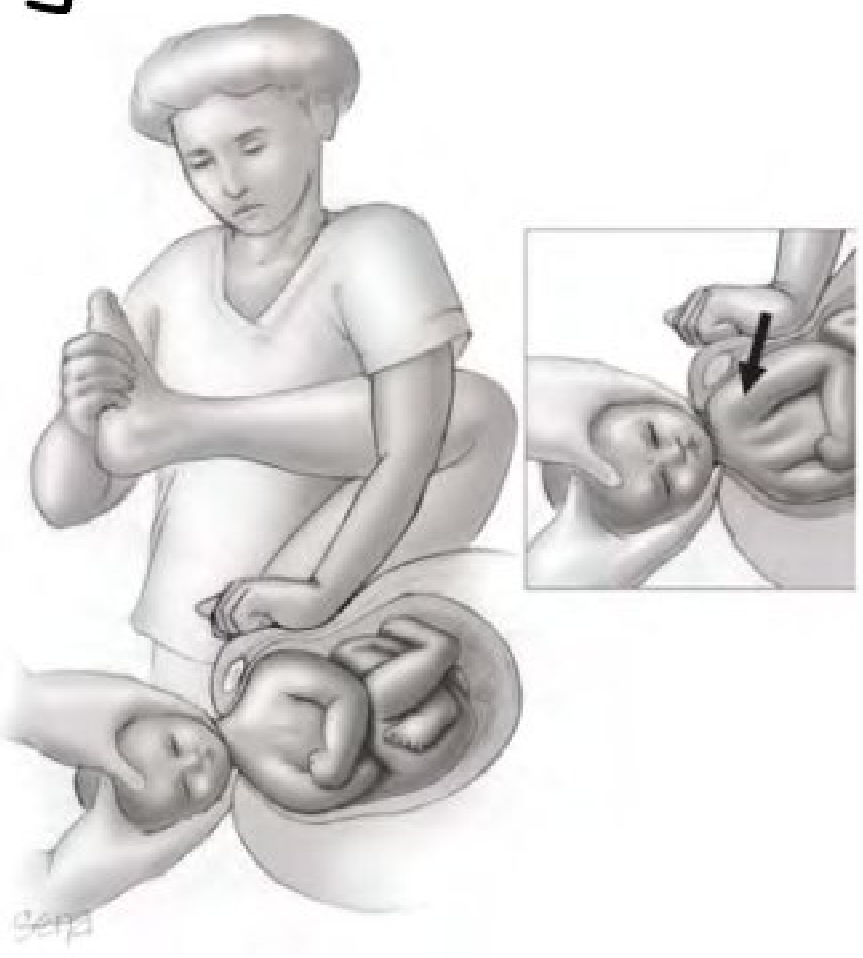
Macrosomia ↓ Shoulder Dystocia

-Management : Shoulder Dystocia Drill

"HELPERR"

H = call for Help
E = Give Episiotomy
L = Flex Legs (i.e do MC Roberts Maneuver)
↳ Later on abduction of Legs



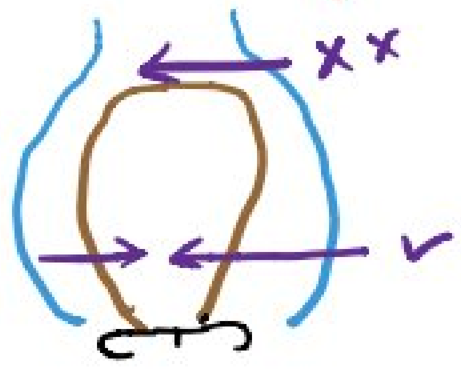


MC Robert's Maneuver

-MC nerve injured during MC Robert's maneuver is : Lateral Femoral cut. N.

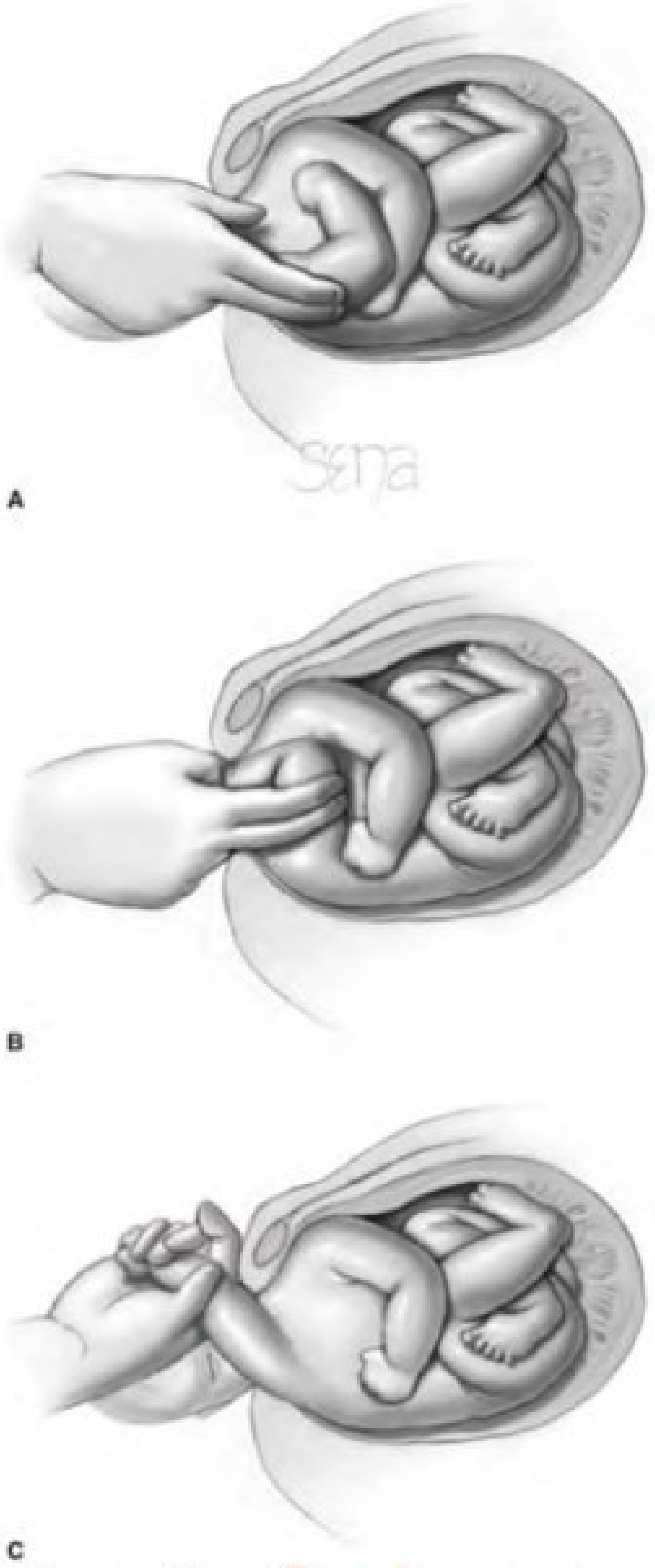
P = Give suprapubic Pressure

# Fundal pressure is C/I



Suprapubic Pressure

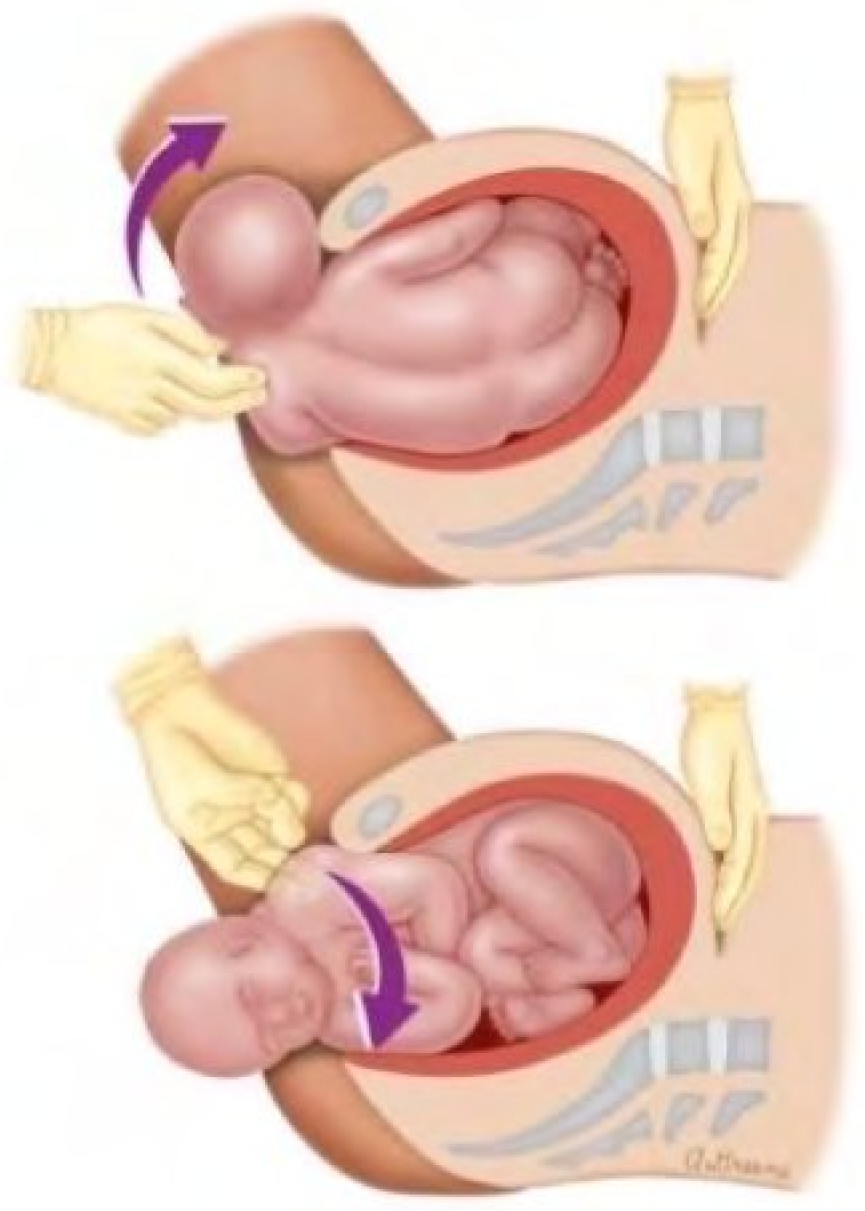
E = Enter vagina = Hold & Rotate shoulder
- Woods corkscrew maneuver
- Rubin maneuver



Woods Cork screw maneuver

FIGURE 27-7

Woods maneuver. The hand is placed behind the posterior shoulder of the fetus. The shoulder is then rotated in a corkscrew manner so that the impacted anterior shoulder is released.



Woods Cork screw maneuver

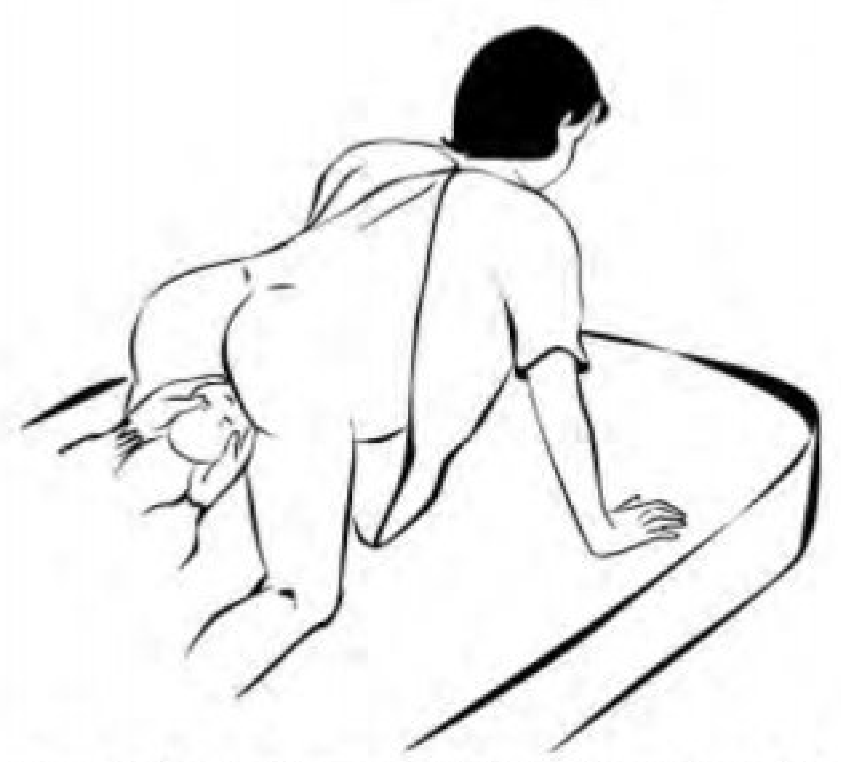


Figure 3. The Gaskin maneuver. Placing the mother in the all-fours position may allow the posterior shoulder to descend. Reprinted with permission from Kovavisarach E. The "all-fours" maneuver for the management of shoulder dystocia. Int J Gynaecol Obstet 2006;95(2):153–4.

Gaskins Maneuver

R= Remove posterior arm k/a
Jacquiemear Maneuver (Barmun maneuver)
Deliver posterior arm of baby
↓
Now it's on one side shoulder & and on another side arm
(Now diameter is acromio auxillary which is 2-3 cm less than Bisacromio diameter)
↓
This helps in shoulder dystocia

R= Rotate patient to all 4 Limbs
Gaskin / all 4 maneuver

- Last maneuver done :
  Zananelli Maneuver → push the head back into uterus f/b cesarean section
- Destructive procedures which are not done (but can be done)
  1. Cleidotomy → # Clavicle
  2. Symphostomy → dividing pubic symphysis in mother

NOTE :

* First maneuver in shoulder dystocia is → Mc Roberts maneuver
* MC maternal complication of shoulder dystocia : PPH
* MC Fetal complication of shoulder dystocia : Brachial plexus injury
* MC Bone injury d/t shoulder dystocia : Clavicle

4. ↑ chances of death of fetus
   - occurs at <20wks / <500 gms in wt = ABORTION (↑)
   - occurs at >20wks / ≥500gms in wt = IUD of fetus (↑)
   - occurs during delivery = STILL BIRTH (↑)

5. Mostly diabetes babies have macrosomia :
   But rarely = IUGR may also be seen <
   → Diabetes + PIH
   → Overt diabetes c̄ vasculopathy

THYROID GLAND

* In pregnancy - undergoes HYPERPLASIA

any visible growth in gland is pathological

* ↑ in TBG → gland will in ↑ T_3 and T_4 (Total)

stimulus is ↑ in HCG

* Free T_3 and T_4 - ↑ (but in Ⓝ Range)

* TSH ↓ compared to non-pregnant state but stays ≤ in Ⓝ Range

* Pregnancy is EUTHYROID STATE

* (MC) cause of HYPOTHYROIDISM in Pregnancy - HASHIMOTO's THYROIDITIS

HYPOTHYROIDISM can cause

1. Abortions
2. Pre-Term Labour
3. LBW Babies
4. Pre-Eclampsia
5. Abruption of Placenta
6. Non-Reassuring FHR

if k/c/o Hypothyroidism pt is on 'x' amount of Thyroxine becomes pregnant then increase dose of Thyroxine by 50%

Iodine RDA in Pregnancy - 250 mcg/day

Iodine excretion ↑sed in Pregnancy

Ⓜ︎Ⓒ cause of Hyperthyroidism : GRAVE'S Disease

Rx:

1st TM - Propylthiouracil

2nd TM/3rd TM - Methimazole

POST PARTUM BLUES / DEPRESSION

- d/t sudden fall of Estrogen and Progesterone
  ↓
  Responsible for 1st stimulus for INITIATION OF LACTATION

PRE-ECLAMPSIA

I. GESTATIONAL HYPERTENSION :

1. BP ≥ 140/90 mmHg on 2 occasions - 4hrs apart
2. HIGH BP - beyond 20wks of GESTATION
3. NO EVIDENCE of Proteinuria or END ORGAN Damage
4. BP return to Ⓝ ≤in 12wks POST PARTUM

II. PRE-ECLAMPSIA

1. BP ≥ 140/90 mmHg on 2 occasions - 4hrs apart
2. HIGH BP - beyond 20wks of GESTATION
3. ≤ EITHER ONE or BOTH - Proteinuria or END ORGAN Damage

4. BP return to Ⓝ ≤in 12wks POST PARTUM

PROTEINURIA * In O.P.D ⟶ DIPSTICK - +2

* 24hr Urine Protein Estimation : ≥0.3g/24hr

* Urine Protein : Creatinine Ratio : >0.3 mg/mg

END ORGAN DAMAGE - any of the following CRITERIA

1. S. Creatinine >1.1
2. Platelet Count < 1 lakh
3. LIVER ENZYMES are more than TWICE the Ⓝ value (**AST ≥ 70 I.U**)
4. Pulmonary Edema
5. Cerebral / Visual Symptoms

PRE-ECLAMPSIA → **MILD**

→ **SEVERE Criteria** : Either ONE/BOTH 1. BP ≥ 160 / 110 mm Hg and/or

2. END ORGAN DAMAGE

CRITERIA which has been removed from SEVERE PRE-ECLAMPSIA

---

1. Proteinuria - ≥5g/24hr
2. IUGR
3. OLIGURIA

} These are **FINDINGS in PRE-ECLAMPSIA**

iii. **ECLAMPSIA** : Pre-Eclampsia + Seizures

iv. **CHRONIC HYPERTENSION in Pregnancy**

1. High B.P even BEFORE Pregnancy
2. High B.P in 1st 20wks of Pregnancy
3. High B.P even after 12wks POST PARTUM

PATHOPHYSIOLOGY :

1. ABSENT Vascular Remodelling
2. ALTERED Immune Response ⟶ To Paternal Antigen
   - (MC) s/i PRIMI GRAVIDA at 1st Exposure
   - ↑ Paternal Antigen; higher will be RISK
     eg: TWIN PREGNANCY
3. GENETIC - 1st degree relative affected then RISK is HIGHER

Ⓝ VASCULAR REMODELLING

Trophoblast which forms VILLUS - **VILLOUS TROPHOBLAST**

EXTRA VILLOUS / INVASIVE TROPHOBLAST

Spiral Artery - **ENDOVASCULAR TROPHOBLAST**

into Decidua - **INTERSTITIAL TROPHOBLAST**

SPIRAL ARTERY
- highly coiled and highly resistant → undergoes **VASODILATION** to **LOW Resistant** → **GOOD UTEROPLACENTAL CIRCULATION**
- Resistant to Vasopressors

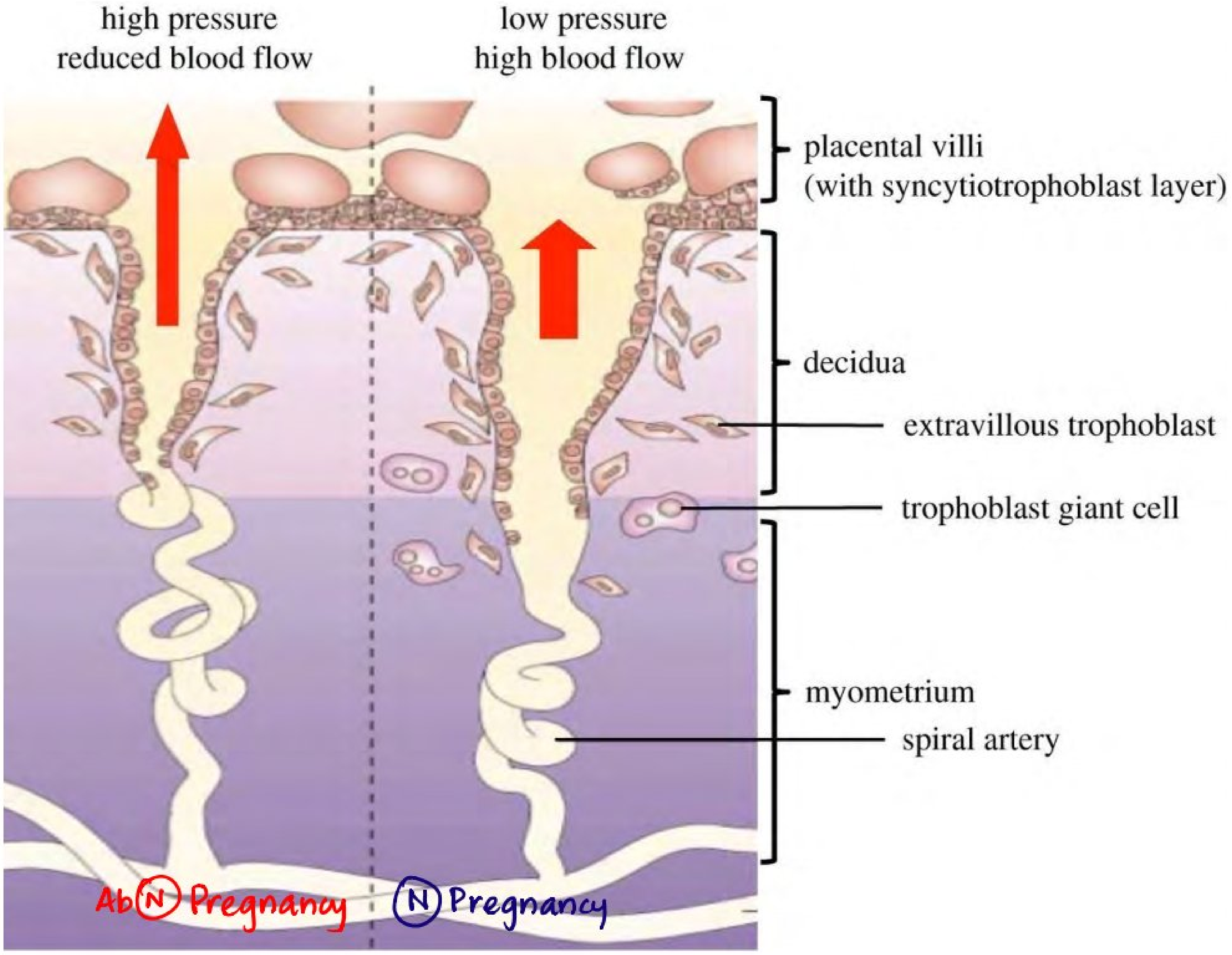
CELLS DOING VASCULAR REMODELLING

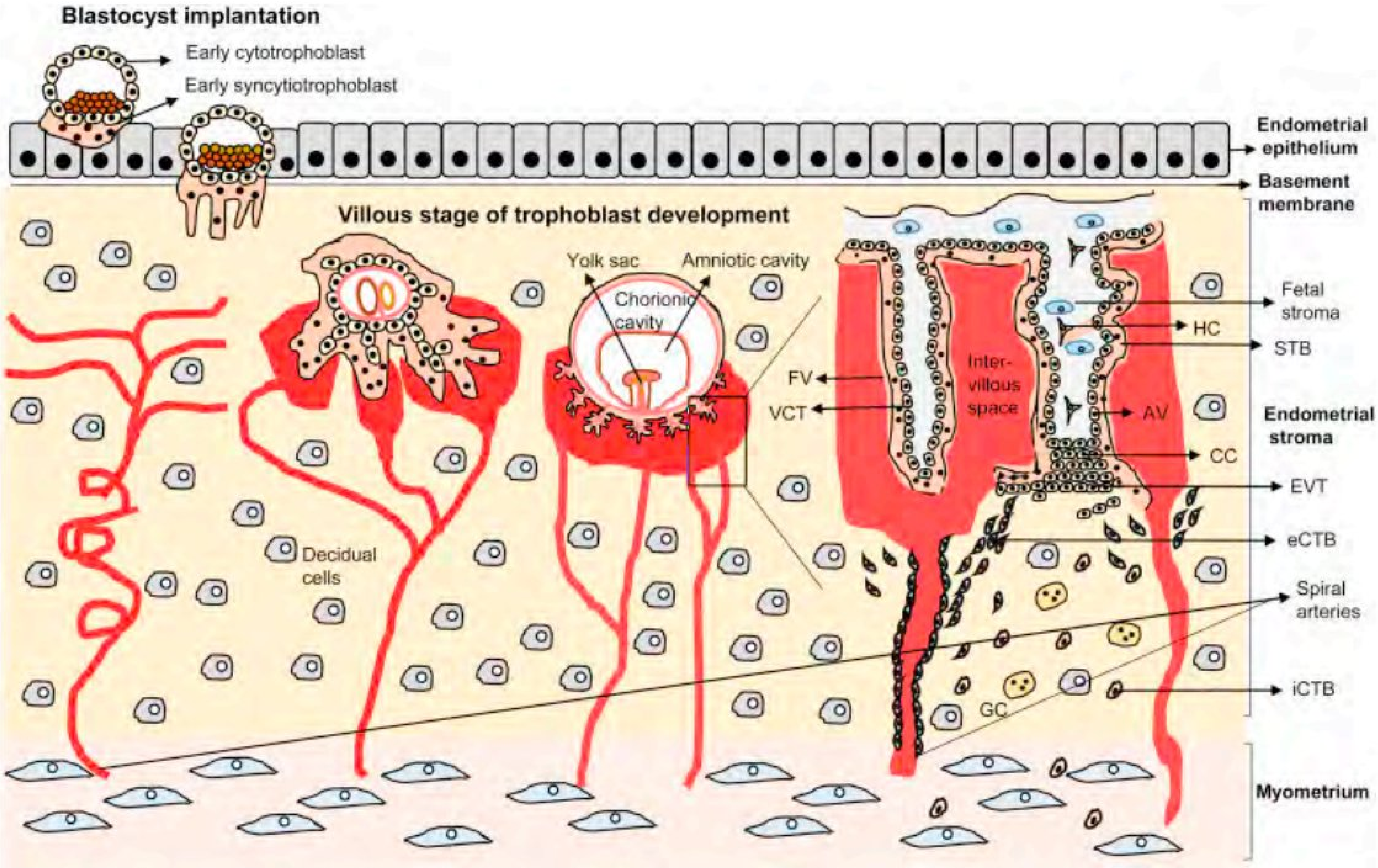
1. **EXTRA VILLOUS, INVASIVE, ENDOVASCULAR Trophoblast**
2. **DECIDUAL NATURAL KILLER Cells** : controls the process

Vascular Remodelling occurs in **2 Phases**

1st Phase - by 12 wks
2nd Phase - by 16 wks

if ABSENT → **UTERO PLACENTAL INSUFFICIENCY**
↓
1. IUGR
2. Pre-Eclampsia
3. Adherent placenta





HIGH RISK FACTOR

1. Previous h/o Pre-Eclampsia
2. Chronic HTN
3. Renal ds
4. Diabetes
5. APLA Syndrome
6. Primi gravida
7. High BMI
8. Twin Pregnancy
9. Molar Pregnancy
10. Extremes of Age ≤18yrs or ≥40yrs

PROTECTIVE

1. Smoking

Pre-Eclampsia has NEGATIVE a/w PLACENTA PRAEVIA

PREVENTION

Indication in HIGH Risk women

1. LOW DOSE ASPIRIN - 80-150mg , started at 12wks ⟶ continue throughout Pregnancy

2. Ca^{2+} Supplementation - has role only in women who are Ca^{2+} deficient pre-conceptionally

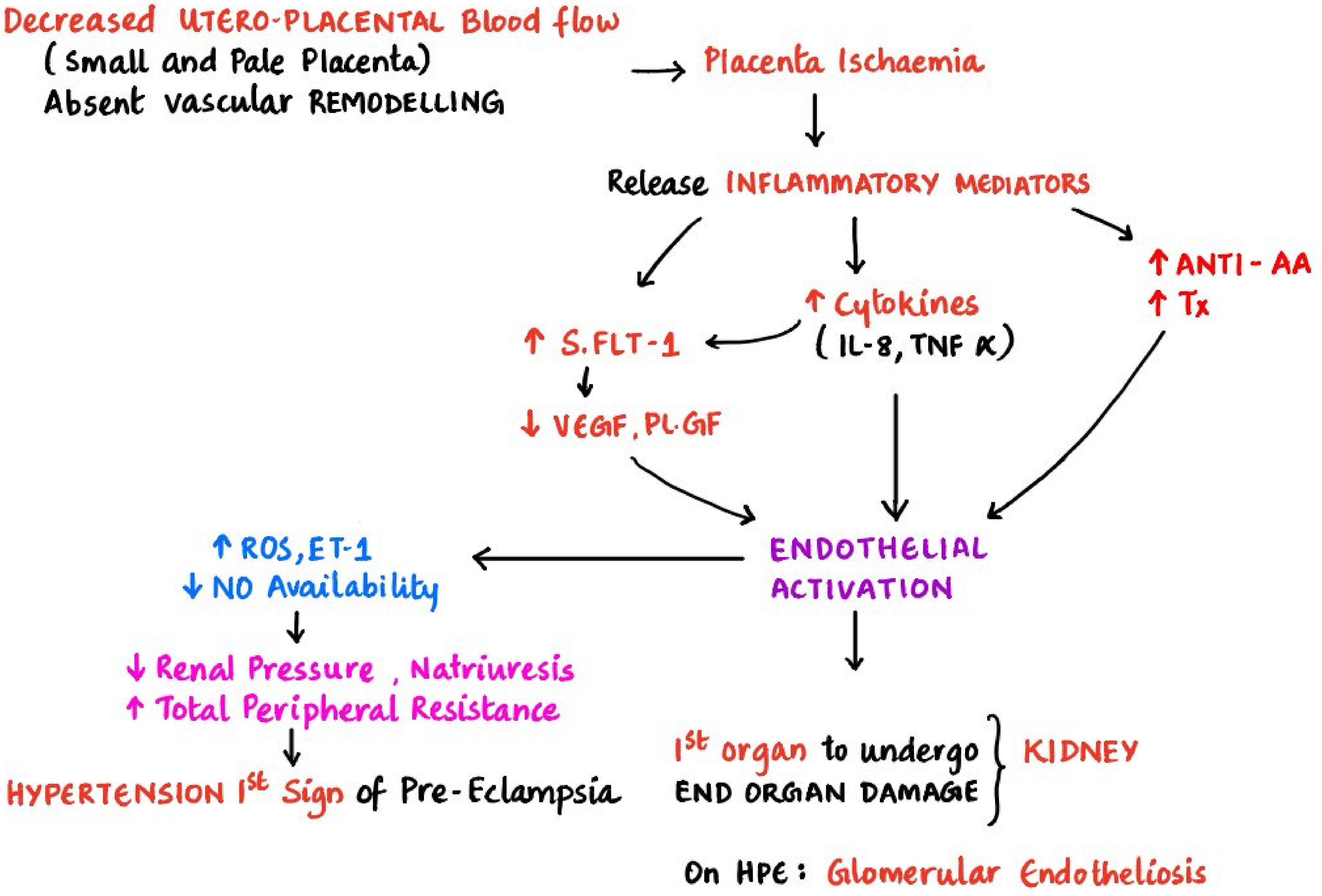
GOI : Ca^{2+} Supplementation ⟶ to meet RDA : 1200 mg
1000mg (500mg BD) started at 12wks

PREDICTION OF PRE-ECLAMPSIA

ANTI-ANGIOGENIC Factors
1. S.FLT-1
2. S.Eng

ANGIOGENIC Factors
1. VEGF
2. Pl.GF

S.FLT 1	Solube Fms Like Tyrosine kinase-1
S.Eng	Soluble Endoglin
VEGF	Vascular Endothelial Growth Factor
Pl.GF	Placental Growth Factor
AT1-AA	Auto Antibodies to Angiotensin II Type I Receptor
Tx	Thromboxane
ROS	Reactive Oxygen Species
ET	Endothelin 1



Decreased UTERO-PLACENTAL Blood flow
(Small and Pale Placenta)
Absent vascular REMODELLING → Placenta Ischaemia

↓

Release INFLAMMATORY MEDIATORS

↑ S.FLT-1 ← ↑ Cytokines (IL-8, TNF α)

↓ VEGF, Pl.GF

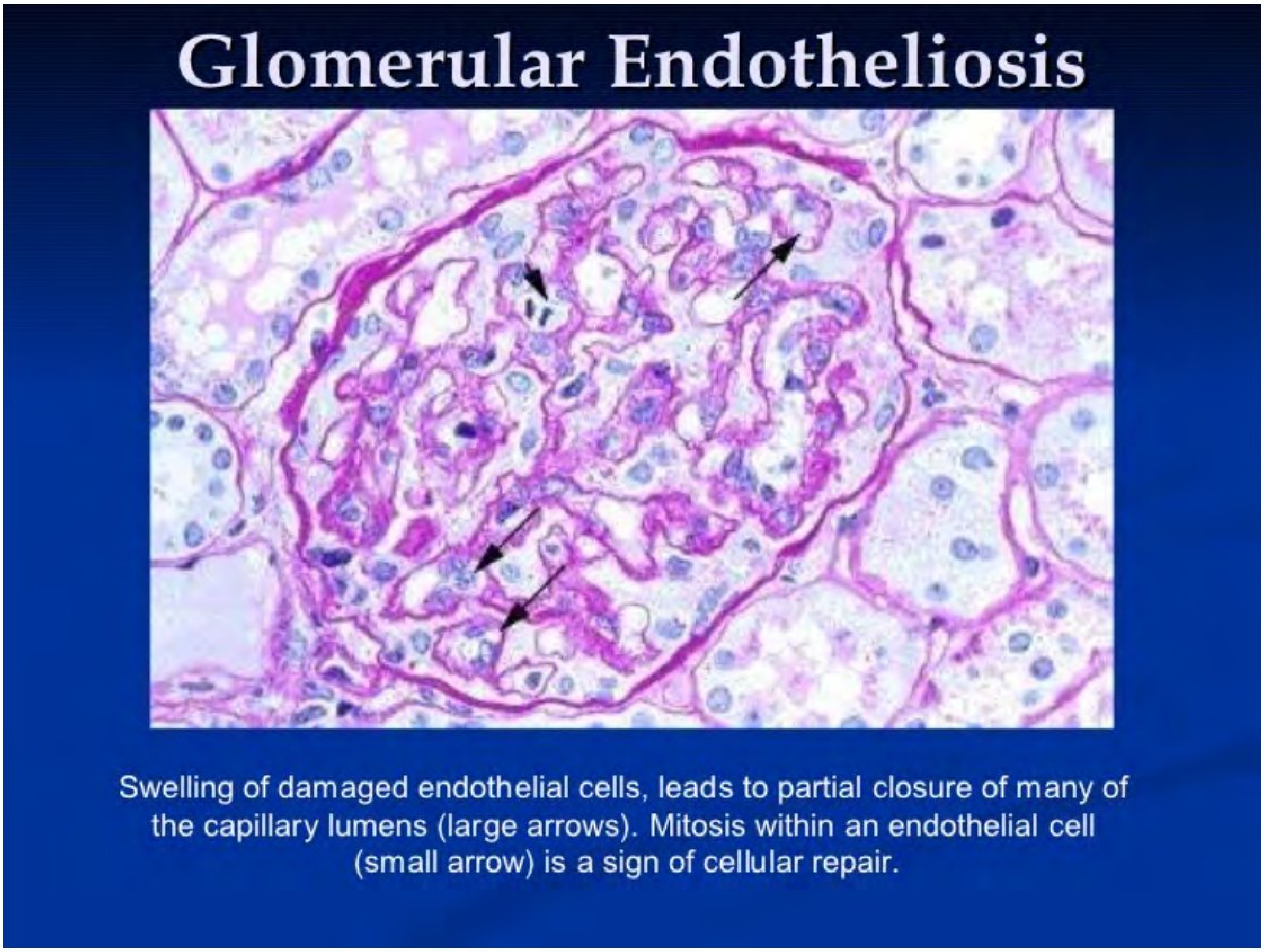
↑ ANTI-AA
↑ Tx

→ ENDOTHELIAL ACTIVATION

↑ ROS, ET-1
↓ NO Availability
↓
↓ Renal Pressure, Natriuresis
↑ Total Peripheral Resistance
↓
HYPERTENSION 1st Sign of Pre-Eclampsia

1st organ to undergo END ORGAN DAMAGE } KIDNEY

On HPE: Glomerular Endotheliosis



Glomerular Endotheliosis

Swelling of damaged endothelial cells, leads to partial closure of many of the capillary lumens (large arrows). Mitosis within an endothelial cell (small arrow) is a sign of cellular repair.

PREDICTORS

① S.FLT-1 - ↑

② S.ENG - ↑

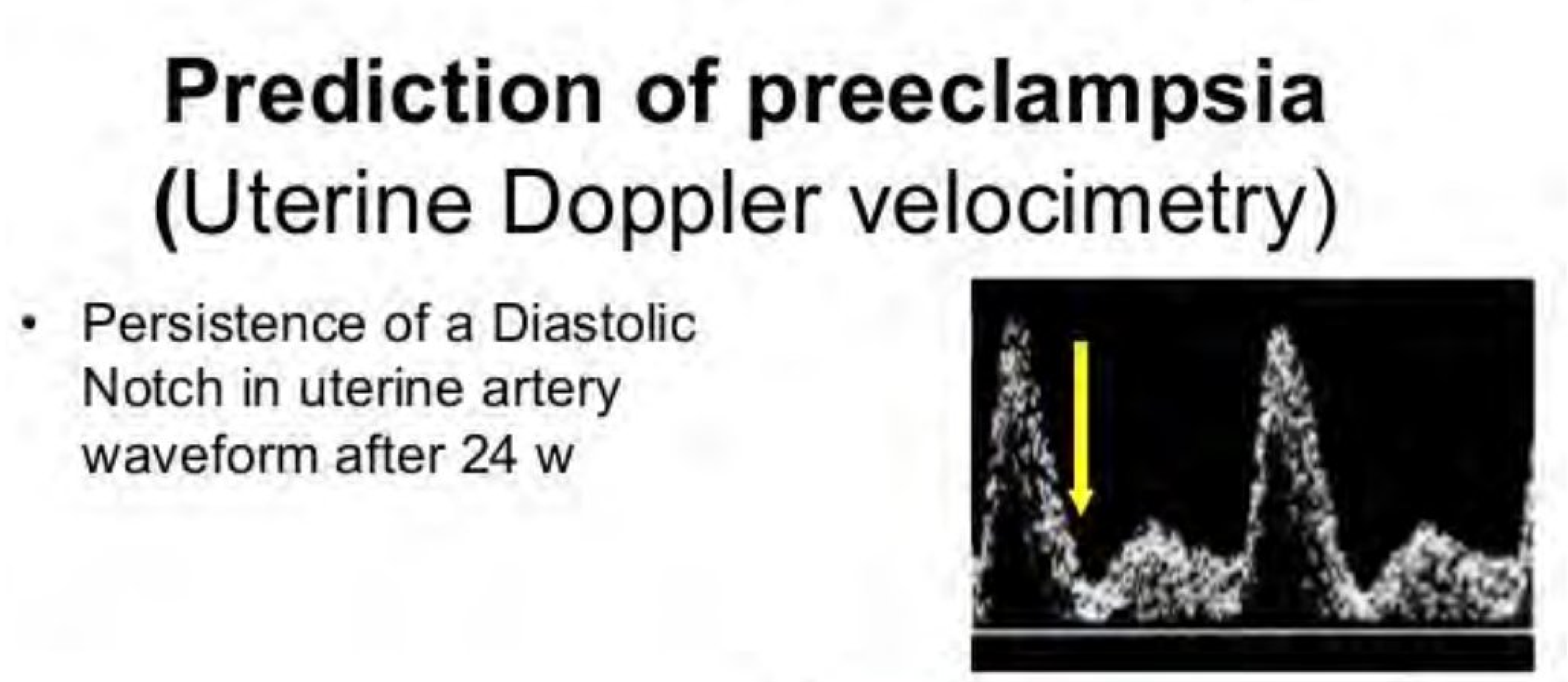
③ PL.GF - ↓

④ VEGF - ↓

⑤ Urinary Ca^{2+} excretion - ↓

⑥ Uterine Artery Doppler Study

Uterine Artery Doppler Study - "NOTCH" → High Resistance Vessel
Persistent Notching >22wks (22-24wks)



Prediction of preeclampsia (Uterine Doppler velocimetry)

- Persistence of a Diastolic Notch in uterine artery waveform after 24 w

Specific Rx of Pre-Eclampsia : Remove the Placenta - TERMINATION OF PREGNANCY

IMPENDING ECLAMPSIA

C/F : 1. Headache and Dizziness - CEREBRAL HYPOXIA

2. Epigastric pain f/b Nausea and Vomiting

3. Visual symptoms : Sudden onset of blurring / diplopia / Scotomas / Blindness

BLINDNESS d/t

1. Retinal detachment
2. Occipital lobe Hypoxia

HYPERTENSIVE RETINOPATHY - KEITH WEGNER's Classification

On Fundus Examination

- papilloedema and silver wiring

Q. Primi 34 wks, headache and blurring of vision x 4hrs. O/E BP 160/100 mm Hg, Protein - 2+, FHR - Ⓝ. Next Step in Mx ?

Ans: Prevent Seizures + Anti-HTN + Termination of Pregnancy

HELLP SYNDROME

- feature of Severe Pre-Eclampsia ; 15% of woman - Ⓝ BP
- End Organ Damaged → LIVER
- (MC) Complication : ABRUPTION
- (MC) s/w MULTIGRAVIDA
- (MC) Presentation : EPIGASTRIC / Ⓡ upper quadrant pain
- (MC) present in 3rd TM

DIAGNOSIS

1. **Hemolysis** - ① On P/s - SHISTOCYTES , HELMET CELLS
   ② S. Total Bilirubin (unconjugated) - >1.2 mg/dl
2. **Elevated Liver enzymes** - ③ AST (SGOT) ≥ 70 IU
3. **Low Platelet count** - ④ Platelet count < 1lakh

FINDINGS : ① ALP ↑
② LDH ↑

Recurrence Risk - upto 7% (4-7%)
Mortality Rate - 2%

Other presentation similar to Pre-Eclampsia

D/D of HELLP

1. AFLP (A/c Fatty Liver of Pregnancy) : (MC) cause of A/c Liver Failure in Pregnancy

- presents in 3rd TM
- Pain
- More Severe form of LIVER injury

Any of the following : AFLP

1. Hypoglycemia
2. Hepato-Renal Syndrome - (rising S. Creatinine)
3. Evidence of COAGULATION FAILURE
4. Pancreatitis

HIGHER Mortality Rate. Confirmation of Dx by POST MORTEM (LIVER BIOPSY)

RISK FACTORS

1. Pre-Eclampsia
2. Multifetal pregnancy
3. Male fetus

Enzyme deficiency a/w AFLP : LCHAD
(Long Chain Hydroxy Acyl Dehydrogenase)

If Fetus - Homozygous → Manifest

2. OBSTETRIC CHOLESTASIS

- presents in 3rd TM as PRURITIS (Palms and Soles)
- Hormone a/w ESTROGEN
- Genetic mutations : ABC B11 (ABC B4)

Diagnostic Test : S. Bile acids

INVESTIGATIONS

1. S. Total Bilirubin (conjugated Hyperbilirubinemia) - ↑ (mildly)
2. Liver findings - Aminotransferases - ↑ , ALP - ↑

Dangerous to Fetus → Prematurity → Ⓜⓒ cause NEONATAL DEATH
→ MAS (Meconium Aspiration Syndrome)
→ Sudden IUD

- Bile acids irritation and cause Pre-mature labour
- Bile acids reach Fetal Gut ⟶ Irritation ⟶ Pass meconium
- IUD is d/t ASPIRATION of THICK MECONIUM

Rx: 1. URSODEOXYCHOLIC ACID - ↑ses Bile excretion

Termination of Pregnancy at 37wks

Ⓜⓒ Hepatitis in Pregnancy ⟶ HEPATITIS-B

Ⓜⓒ A/c VIRAL HEPATITIS in Pregnancy ⟶ HEPATITIS-E (Highest Mortality Rate)

MANAGEMENT OF PRE-ECLAMPSIA

NON-SPECIFIC (SYMPTOMATIC) TREATMENT

- Control BP
- Mx of CONVULSIONS

} These DONOT STOP PROGRESSION of disease

- This Rx CANNOT REVERSE the process
- REDUCES Morbidity and Mortality Rates

SPECIFIC TREATMENT

1. STOPS the progression of ds.
2. REVERSE the process
3. Removal of Placenta
4. Termination of Pregnancy

HIGH B.P - when to start drug (INDICATIONS)

1. If B.P persistently ≥ 150/100 mmHg (but < 160/110 mmHg)

DOC for Pre-Eclampsia
HTN in Pregnancy
} LABETALOL ($\alpha + \beta$ Blocker)

2. If B.P ≥ 160/110 (A/c HTN in Pregnancy)

i) DOC : iv LABETALOL, start ≤ 20mg i.v $\xrightarrow[\text{if still elevated}]{\text{10min;}}$ 40mg $\xrightarrow[\text{if still elevated}]{\text{10min}}$ 80mg $\xrightarrow[\text{if still elevated}]{\text{10min}}$ 80mg

Maximum dose of LABETALOL given in a day = 300mg

ii) i.v HYDRALAZINE : begin ≤ 5mg $\xrightarrow{\text{INCREASE}}$ 10mg

iii) **NIFEDIPINE** (Sustained Release ORAL Tab)

iv) **i.v NTG** : given if Pulmonary Edema

v) **SODIUM NITROPRUSSIDE** : Drug of LAST RESORT (used in REFRACTORY HTN)

↳ because it cause CYANIDE TOXICITY

CHRONIC HYPERTENSION IN PREGNANCY

SAFEST Anti-HYPERTENSIVE : METHYLDOPA > LABETALOL

↓

- Not given in A/c HTN in Pregnancy because → DELAY in ONSET of Action
- MILD ANTI-HYPERTENSIVE

ANTI HYPERTENSIVES C/I IN PREGNANCY

TARGET BP :
SBP - 120-140 mmHg
DBP - 80 - 100 mmHg

1. ACE inhibitors
2. ARB
3. BETA-BLOCKER } can be given if there is a Cardiac indication
4. DIURETICS } UTEROPLACENTAL INSUFFICIENCY further ↓ INTRA-VASCULAR VOLUME - ↓
5. DIAZOXIDE - K^+ channel ACTIVATOR

TERMINATION OF PREGNANCY

1. GESTATIONAL HTN
2. MILD PRE-ECLAMPSIA

} Well Controlled → Termination of Pregnancy at 37wks

VAGINAL Delivery PREFERRED

3. SEVERE PRE-ECLAMPSIA - Well controlled → Termination of Pregnancy at 34wks

VAGINAL Delivery PREFERRED

TERMINATION OF PREGNANCY (IRRESPECTIVE OF POG)

1. IMPENDING ECLAMPSIA
2. ECLAMPSIA
3. HELLP Syndrome
4. FETAL DISTRESS / ABRUPTION
5. Uncontrolled B.P
6. Rising S. Creatinine

DOPPLER : is used for Fetal Well being. (MC) used vessel : is UMBILICAL ARTERY

↑ It is a LOW RESISTANCE VESSEL

1. In Utero-placental Insufficiency → $\frac{\text{Blood flow SYSTOLE}}{\text{Blood flow DIASTOLE}}$ ⇒ S/D Ratio : ↑
(UPI)

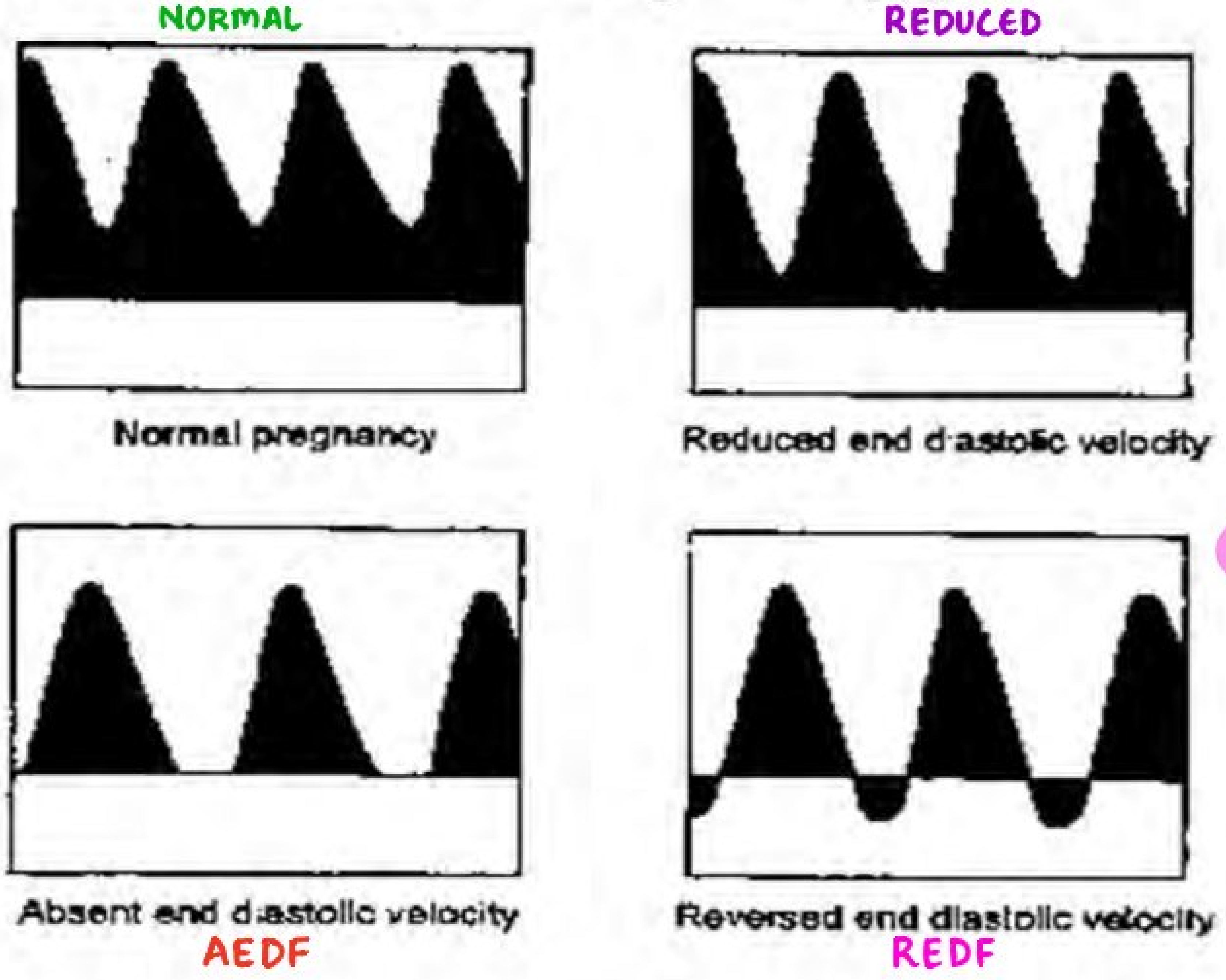
In Ⓝ Pregnancy as POG ↑ - S/D Ratio ↓

S/D Ratio ≥ 3 @ 28wks is MARKER OF UPI

2. AEDF (ABSENT End Diastolic Flow)

3. REDF (REVERSAL of End Diastolic Flow)

Umbilical Artery Doppler


NORMAL
REDUCED
Normal pregnancy
Reduced end diastolic velocity
Absent end diastolic velocity
AEDF
Reversed end diastolic velocity
REDF

MANAGEMENT OF

1. AEDF
   @ ≥ 34wks → T.O.P by C.S
   @ < 34wks → STEROIDS ↓ Inj. DEXAMETHASONE
   - 6mg i.m 4 doses 12hrs apart

   for lung maturity → T.O.P by C.S

   Continue daily Fetal Monitoring

2. REDF : if Pregnancy
   < 32wks - Steroids → Monitor Fetus → T.O.P by C.S
   ≥ 32wks - T.O.P by C.S

ECLAMPSIA

Pre-Eclampsia + Seizures GTCS (Grand Mal)
↳
① CEREBRAL HYPOXIA
② CEREBRAL EDEMA

(MC) → ANTEPARTUM - 1st Seizure BEFORE LABOUR
INTRAPARTUM - 1st Seizure DURING LABOUR
(LC) → POSTPARTUM - 1st seizure ≤in 48hrs

Q. Pt. on wheel chair - 8½ wks Pregnancy - 1st seizure @ home. Now presents ≤ Seizure. Mx ?

Ans: Stabilize the position of patient ⟶ CIRCULATION : Check PR - 114/min
BP - 160/110 mm Hg
↓
AIRWAY : Mouth gag ; Suction out any secretions
O$_2$ by mask.
↓
- for prevention of Recurrence of sei ure

Inj. $MgSO_4$

1. Central action : NMDA Receptors ; Cerebral vasodilation
2. Blocks Ca^{2+} Channels in Periphery @ High doses
3. NO Anti HTN Effect, So separate drug given. DOC: LABETALOL

$MgSO_4$ [PRITCHARD'S REGIME]

Loading dose $\xrightarrow{\text{after 4hrs}}$ MAINTENANCE dose → Given every 4hrly till 24hrs after delivery (or) 24hrs after last seizure (whichever is LATER)

LOADING DOSE - 14g

i.e 4g i.v + 10g i.m → given as divided dose 5g i.m in EACH BUTTOCK

↳ Slow iv preferably over 10min (Monitor HR)

For i.v → 20% $MgSO_4$

For i.m → 50% $MgSO_4$

MAINTENANCE DOSE

Before Starting Check for Mg^{2+} toxicity :

Toxicity present if any one of the 3 following

1. Patellar Reflex ⊕
2. Resp. Rate - >12/min
3. Urine output - ≥100ml/4hrs

} Give 5g 50% $MgSO_4$ i.m

[Main Route of Mg^{2+} excretion in urine]

So, OLIGURIA can cause Mg^{2+} toxicity

Therapeutic levels of Mg^{2+} = 4-7 meq

ABSENT DTR (Patellar Reflex Sluggish) - 10 meq

RESPIRATORY DISTRESS - 12 meq

RESPIRATORY ARREST - 15 meq

CARDIAC SYMPTOMS - 20-25 meq

If any of the 3 Parameters ABNORMAL

↓

Omit $MgSO_4$ and Send Blood Sample for S. Mg^{2+} lvls

↓

if elevated give ANTIDOTE

10ml of 10% i.v Ca^{2+} Gluconate

SPECIFIC TREATMENT : T.O.P by vaginal delivery is PREFERRED provided if we can deliver her c̄in 24hrs of 1st Seizure or else → C.S done under G.A

Preferred Anesthesia for CS in PRE-ECLAMPSIA : EPIDURAL ANESTHESIA

PREVENTION OF RECURRENCE (Rx of ECLAMPSIA → $MgSO_4$)

PROPHYLACTIC $MgSO_4$ given in 1. IMPENDING ECLAMPSIA

2. HELLP Syndrome

$MgSO_4$ ⟶ NEUROPROTECTION if anticipating delivery < 32wks (Pre-Term)

$MgSO_4$ @ 9 meq or more ⟶ TOCOLYTIC ACTION ⟶ So, not used d/t TOXICITY

Rh NEGATIVE PREGNANCY

Husband Blood Grp ⊕ ve ⟶ Sensitization : Feto-Maternal hemorrhage

1. At the time of delivery - (MC)
2. In 3rd TM - 2nd (MC)

Minimum amount that can cause SENSITIZATION is 0.1 ml

- 1st Pregnancy is not affected because 1st produces IgM Ab (these donot cross placenta)
- For Sensitization : ICT [Indirect Coombs Test] done if Husbands Blood grp is ⊕ve

- If ICT ⊖ve ∴ Not sensitised
  - Prevent by giving Anti-D (given for prophylaxis)
  - If we suspect FMH < 12wks = dose 50mcg
  - >12wks = 300mcg - protect from 30ml of fetal blood o 15ml of fetal RBC
  - if >30ml = KB Test. Calculate the dose of Anti-D

- If ICT ⊕ve : Sensitive ; no Role of Anti-D

Titre Critical ≥1:16 → Significant Fetal anemia to cause Heart failure

Test done : MCA Doppler [Peak Systolic Velocity] ≥ 1.5 MoM - Significant Anemia → do CORDOCENTESIS → to know exact Hb

- If fetal Hb ≤ 5g % - Hb HYDROPS FETALIS

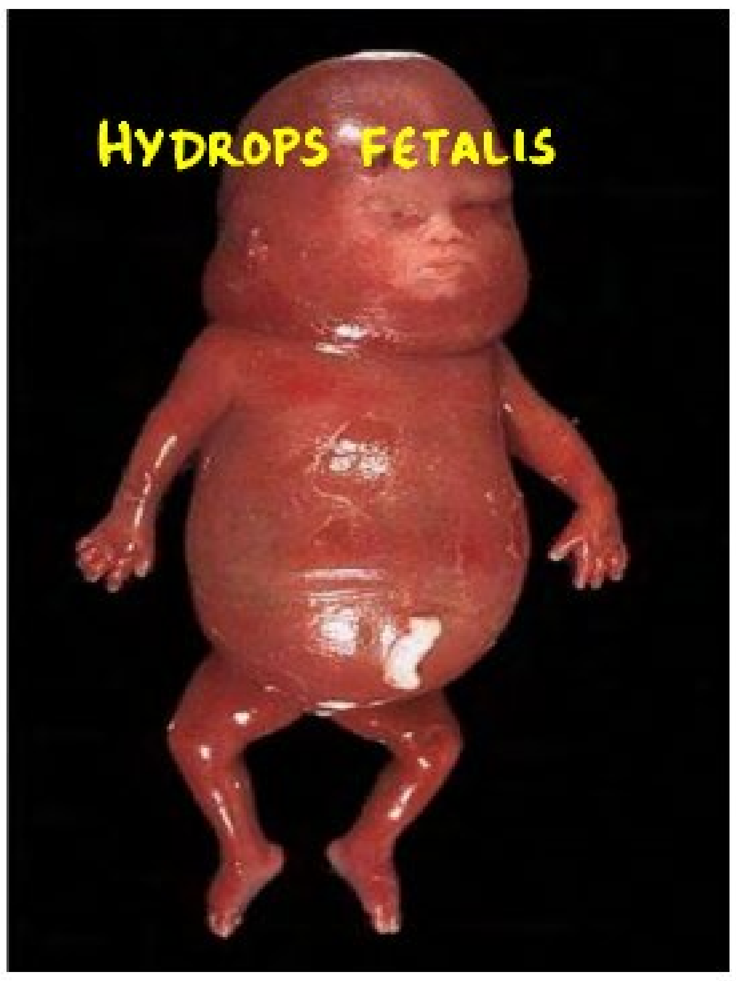
Diagnosis of HYDROPS FETALIS by USG if ≥ 2 of the following :

1. Pericardial Effusion
2. Pleural Effusion
3. Ascites
4. S/c Edema : Halo around the head → "BUDDHA Sign"

Placentomegaly ; Polyhydraminos

- (MC) cause HYDROPS FETALIS - Rh iso immunisation

- (MC) cause Non-Immune HYDROPS FETALIS : CVS Abnormality
  ↳ in Asians : α-Thalassemia
  viral infection : PARVO VIRUS B-19



1st SCENARIO :

NOT SENSITISED

For Rh⊖ve pt

1st Invg : Husbands Blood grp → ICT⊖ → 1st ANC 8wk ICT⊖ → Repeat ICT at 28wk ICT ⊖ve → Give Anti D 300mcg i.m

↓

delver and check if Babies Blood Group is ⊖ve ? → if Baby Bld Grp is ⊕ve, give 2nd Anti-D 300mcg is given i.m c̄in 72hrs of delivery can be given upto 28days

2nd SCENARIO : If pt is Rh⊖,
Husband Bld Grp ⊕ve;
ICT⊕ve at 28wks

} Titer <1:16 → Repeat ICT x 4wkly (<1:16) → Deliver

3rd SCENARIO :

Rh ⊖ ve
Husband Bld Grp ⊕
ICT ⊕ ≥ 1:16

} do MCA doppler
- < 1.5 Mom → f/u c̄ MCA Doppler
- ≥ 1.5 mom → Significant Anemia
  - ↙ can cause Heart failure so → if ≥ 34wk → Deliver
  - ↘ < 34wk → Give Steroids do USG
    - ↓ Anemia → do CORDOCENTESIS
      - if Hb > 8g% → Repeat Cordocentesis
      - if Hb > 8g% → Intra Uterine Blood Transfusion

Jaundice In Pregnancy

- When the serum bilirubin exceeds 2mg%. (Normal being 0.2-0.8%) visible yellow staining of the tissue appear.
- Incidence : In India is 1-4 per 1000 deliveries

CAUSES OF JAUNDICE DURING PREGNANCY

Jaundice peculiar to the pregnant state

- ∗ Intrahepatic cholestasis (Obstetric hepatosis)- may be recurrent.
- ∗ Severe Pre-eclampsia, Eclampsia, HELLP Syndrome.
- ∗ Acute Fatty Liver (acute yellow atrophy of the liver)
- ∗ Severe hyperemesis gravidarum
- ∗ Endotoxic shock : Disseminated Intravascular Coagulation (DIC)

Jaundice unrelated to Pregnant state

- ∗ Viral hepatitis : Virus A-E, G
- ∗ Gall stone : Obstructive Jaundice
- ∗ Drug induced : Isoniazide, Phenothiazines
- ∗ Hemolytic Jaundice : Mismatched blood transfusion
  Malaria
  Clostridium welchii infection etc

Jaundice when pregnancy is superimposed on chronic Liver disease

- ∗ Chronic Hepatitis
- ∗ Cirrhosis, tumors

VIRAL HEPATITIS

i. Viral hepatitis commonest cause of Jaundice in pregnancy.

ii. Six different types of highly contagious hepatitis virus have been identified. Each type has different clinical effect to the pregnancy women & her fetus

Hepatitis A (HAV)	Hepatitis B (HBV)	Hepatitis C (HCV)	Hepatitis D (HDV)	Hepatitis E (HEV)	Hepatitis G (HGV)
MCC of	MCC of	MCC of		MCC of	

Epidemic in children in India	Vertical (Perinatal) Transmission	BT induced Hepatitis MCC of Liver transplantation in World		Epidemic in India Maximum Risk of maternal mortality & Hepatic Encephalopathy	
TYPES					
RNA	DNA	RNA	RNA	RNA	RNA
INCUBATION (DAYS)					
15-45 Mean : 30	30-180 Mean : 60-90	15-160 Mean : 50	30-180 Mean : 60-90	14-60 Mean : 40	
ONSET					
Acute	Insidious/Acute	Insidious	Insidious/Acute	Acute	
AGE PREFERENCE					
Children Young Adults	Young Adults (Sexual & Percutaneous), babies, Toddlers	Any age, but more common in Adults	Any age (Similar to HBV)	Young adult (20-40yrs)	
ROUTE					
Feco-oral	Parenteral Sexual Vertical Breast milk all other route	Parenteral Faecal-oral Route	a/w HBV either co (or) Super inf.	Behaves like HAV. Leads to Fulminant Hepatitis. Maternal Mortality ↓ 15-20%	Related to HCV Coinfection c̄ HAV, HBV HCV & HIV is common

CLINICAL COURSE

SEVERITY					
Mild	Occasionally Severe	Moderate	Occasionally Severe	Mild	
FULMINANT					
0.1%	0.1-1%	0.1%	5-20%	1-2%	
PROGRESSION TO CHRONICITY					
None	Occasional (1-10%) (90% of neonates)	Common	MCC of Chronicity (85%)	None	
CARRIER					
None	0.1-30%	1.5-3.2% (MC Carrier Stage)	Variable	None	
CANCER					
None	++(neonatal infection)	+	±	None	
PROGNOSIS					
Excellent	Worse c̄ age, Debility	Moderate	Acute, good Chronic, Poor	Good	
PROPHYLAXIS					
IG Inactivated vaccine	HBIg Recombinant vaccine	None	HBV Vaccine (none for HBV Carriers)	Vaccine	
THERAPY					
None	· Interferon · Lamivudine · Adefovir · Pegylated interferon	Pegylated Interferon plus Ribavirin	Interferon ±	None	

	·Entecavir ·Telbivudine				
Mild	Occasionally Severe	Moderate	Occasionally severe	Mild	
DIAGNOSIS					
IgM Ab to HAV	Active Stage (SEC) ·Highly infective ↑sed liver enzymes & HBS Ag +ve HBE Ag +ve IgM HBC Ab +ve ·Low infective HBS Ag +ve Anti HBE Ab +ve IgM HBC Ab +ve ·Chronic carrier (CES) Highly infective HBS Ag +ve (>6 m) HBE Ag +ve IgG HBC Ab +ve Low infective HBS Ag +ve Anti HBE Ab +ve IgG HBC Ab +ve	HCV Ab (EIA) Confirm ↓ RIBA-3		ELISA Anti HEV (IgG & IgM Ab)	
PERINATAL TRANSMISSION					
Rare	1st trim: 10% 3rd trim: 90% Women c̄ HBS Ag &	10-40% (↑↑ when Coinfected c̄ HIV & HBV	Seen	Uncommon	Seen

	HBE Ag +ve -90%				
NEONATAL TRANSMISSION					
Rare	Seen	Seen	Seen	Seen	Seen

UTI In Pregnancy

1. The MC infecting micro-organism : Esherichia coli (90%)
2. Asymptomatic Bacteriuria : Refers to persistent actively multiplying bacteria c̄ in Urinary tract in women who have no symptoms.
3. A clean voided specimen containing >100,000 organism per milliliter is diagnostic. It may be prudent to treat when lower concentration are identified, because pyelonephritis develops in some women c̄ colony counts of 20,000-50,000 organism/ml
4. A single episode of Asymptomatic Bacteriurian can cause Acute pyelonephritis in 25-40% cases
5. ACUTE PYELONEPHRITIS CAN CAUSE
    - a) IUGR
    - b) Preterm labor
    - c) Anemia
    - d) ↑ risk of PIH
6. Cranberry fruit juice is known to prevent recurrences of UTI. It prevents the adhesions of pillin of E.coli to uroepithelium.
7. Nitrofurantoin : DOC for prophylaxis of Recurrent UTI In pregnancy

Hepatitis B

a. 90-95% of patient clears the infection & has full Recovery

b. 1% develops Fulminant Hepatitis → Massive hepatic Necrosis

c. 10-15% becomes chronic & 10% of chronic suffers from chronic active hepatitis, cirrhosis & Hepatocellular carcinoma.

d. Screening : done in all pregnant women @ 1st ANC & Repeated during 3rd trimester & in high risk group (i/v drug abuser, sexual promiscuity, Hemophilliacs, pt on hemodialysis or having multiple sex partners.)

e. Transmitted by
- Percutaneous inoculation
- Sexual intercourse
- Vertical transmission from mother to child.

f. 90% of HBe Ag +ve mother }
10% of anti HBe +ve mother } Transmit HBV inf. to offspring

g. As per WHO Report, In Asia there is 20-40% chance of vertical transmission in HBeAg negative mothers.

h. When the women are infected during
1st trimester : Neonatal infection 10%
2nd trimester : Neonatal infection 10%
3rd trimester : Neonatal infection 90%
} Virtually all are infected in Perinatal period

i. Most Infection occurs approximately at the time of delivery & are not related to breast feeding

j. Serum, saliva & semen are most infectious for Hep B transmission
Hep B Ag is present in all body secretions except Stools.

k. The risk of chronic Hep B inf. in neonates who doesnot recieve immunoprophylaxis & vaccination is 40%.

l. To reduce perinatal transmission (transplacental)
* Lamivudine (150mg/day from 34 wks)
* HBIG

m. MARKER FOR HEP. B

i. HBs Ag : +ve In Acute, chronic & Carrier state
ii. HBe Ag : +ve in Acute, Marker of infectivity
iii. Anti HBs Ab : +ve in Immune stage
iv. Anti HBe Ab : denotes low infectivity
v. Anti HBc Ab (IgM) : +ve in acute stage
vi. Anti HBc Ab (IgG) : +ve in chronic stage

MATERNAL & FETAL MORTALITY IN HEPATITIS

a. Maternal :
- Fatigue is the MC Symptom
- Increased incidence of PPH, Hepatic coma, Renal Failure, Coagulopathy, infection & hepatorenal syndrome.
- Fulminant Hepatitis is seen c̄ HEV > HCV > HAV c̄ mortality 15-2%.

b. Fetal :
- Increased incidence of abortion, PL, IUD But no GCMF Seen.

OBSTETRIC CHOLESTASIS

Intrahepatic Cholestasis of Pregnancy (IHCP) = Icterus gravidarum = Obstetric cholestasis = Cholestatic Jaundice of Pregnancy = Obstetric Hepatosis

- 10-100 fold ↑ in bile acids (Cholic/deoxycholic acid)
- Pruritis is MC presenting feature
- Onset : after 30 wks of pregnancy
- Serum bilirubin Rarely exceeds 5mg/dl
- Serum transaminase are normal to moderately elevated (Seldom exceeds 250U/L)
- Biopsy : Centrilobular bile staining c̄ bile plugs in canaliculi
- Complications :
  - * Preterm labor
  - * PPH, IUFD & MSAF (Meconium stained amniotic fluid)
- Recurrence in future pregnancy is very common.

TREATMENT

- Antihistamines & Emollients
- Vitamin K
- Ursodeoxycholic acid : DOC (↓ pruritis by ↑ bile acid concentration in blood & it also improves biochemical abnormalities)
- CORTICOSTEROIDS
  Dexamethasone (Dose of 12 mg/d for 1 week)
  ↳ Improves biochemical abnormalities } ∴ Less effective
  Doesnot improve pruritis } than UDCA
- Cholestyramine :
  - poor compliance
  - It may be effective in 50-70% of cases.
  - Causes
    Vit K deficiency → Fetal coagulopathy, Intra crania hemorrhage & Still Birth.

OBSTETRIC MANAGEMENT

i. Pt's of IHCP → ↑ perinatal mortality
↓
∴ fetal surveillance done c̄ Biweekly NST

ii. Conventional antepartum testing, doesnot predict fetal mortality as there is sudden death in cholestasis d/t acute hypoxia Hence delivery Recommended at 37-38 wks.

iii. In those patients c̄ Jaundice (S.bilirubin >1.8mg%), termination of pregnancy should be done at 36 weeks

INFECTIONS IN PREGNANCY

- MC : CMV
- Most teratogenic : Rubella
- MC time for Rubella transmission to fetus : 1st trimester
  (maximum 1-4 wks)
  Absent transmission : beyond 20 weeks
- In Rubella MC Single defect which occurs : Sensorineural HL
- Heart defects seen in Rubella : Patent ductus arteriosus &

Pulmonary Artery Stenosis

- After Rubella vaccine, Pregnancy C/I for 1 month
- CMV Transmission : can occur any trimester
- Most severe infection occurs if transmission occurs in 2nd trimester
- CMV transmission can occurs during Vaginal Delivery & Breast feeding also
- Primary Infection – leads to 40% transmission
- Recurrent Infection – leads to 0.2-2% transmission
- CMV Never leads to Heart defects in fetus
- MC time for toxoplasma infection : 3rd Trimester
- Fetus is affected maximum/ most severly if fetal infection occurs in 1st trimester.
- Triads of Toxoplasma Infection : Intracerebral Calcification
  Chorioretinitis
  Hydrocephalus
- Treatment : SPIRAMYCIN
  ↳ Prevents fetal transmission
  Can't prevent fetal infection if present.

Spiramycin + Pyrimethamine & sulfonamide combination
↳ To treat fetal infection & prevent further transmission

DRUG OF CHOICES

Infections	DOC In Pregnancy
Bacterial Vaginosis	Metronidazole to pt only 1st trimester - Clindamycin
Pneumocystis carinii.	Sulfamethazole - trimethoprim
Typhoid	3rd gen Cephalosporins / Azithromycin
Syphilis <1 yr >1yr	Benzathine pencillin 2.4 million U. i.m single dose Benzathine pencillin 2.4 million U. i.m weekly x 3
Gonorrhea	Inj. ceftriaxone 125 mg i.m single dose or Tab Cefixime 400 mg single dose or inj. spectinomycin 2 gm i.m single dose

Infections	DOC In Pregnancy
Chlamydia	Azithromycin single dose or amoxicillin 500mg TDS x 7days, 2nd choice - Erythromycin
Group B Streptococci	Pencillin, 2nd Best: Ampicillin. In pt who are pencillin resistant - Cefazolin
Malaria- Prophylaxis Treatment	Chloroquine Chloroquine. For radical cure Premaquene is advised after delivery
Appendicitis	Immediate appendicectomy
Red degeneration	Conservative management No termination of pregnancy No myomectomy

DRUG & TERATOGENICITY

Drug	Teratogenicity
Alcohol	Fetal Alcohol Syndrome Pre & Post natal growth Restriction Facial abnormalities : Shortened palpebral fissures, Low set ears, smooth philtrum, thinned upper lip & midfacial hypoplasia CNS defects : Microcephaly, Mental Retardation & Behavioural Disorders
Warfarin	Fetal Warfarin syndrome Chondrodysplasia punctata, Epiphyseal stippling, Nasal Hypoplasia, Optic atrophy & Microcephaly
Phenytoin	Fetal Hydantoin Syndrome Hypertelorism, Broad nasal bridge, low set ears, hypoplastic nails & digitis

Drug	Teratogenicity
Sodium Valproate	Fetal valproate Syndrome Brachycephaly c̄ high forehead, hypertelorism, small nose & mouth, shallow orbits, overlapping fingers & toes and hyperconvex nails.
Diethyl Stilboestrol	Neural Tube defectas Vaginal & cervical adenosis, clear cell adenocarcinoma, Uterine anomalies, Cryptorchidism & testicular hypoplasia
Isotretinoin	Cleft palate, NT defects, Microcephaly, Deafness, Blindness Cardiac defects like atrial septal defects & great vessel defects
ACE ⊖	Renal Tubular dysgenesis, Anuria, Oligohydramnios
Androgen & Danazol	Masculinisation of a female fetus.
Antineoplastic Agents	IUGR, Craniosynostosis, Micrognathia, Severe limb abnormalities.
Tetracyclines	Discolouration of Deciduous teeth.
Cocaine	Microcephaly, Limb Reduction Defect & Genitourinary malformation d/t Cerebral infarction.

NOTE: CNS Anomaly (NTD) + CVS Anomaly + Facial defects

↓

Seen c̄ use of Isotretinoin

Food & Drug Administration (FDA) charted out categories for drugs, taking into account the possible fetal advese effects

Category A : Drugs which have no fetal risks as demonstrated by well controlled studies in human

Eg: Pencillin, Ampicillin

Category B : Drugs which have shown no risk in animal studies but human, studies does not exist, or also if any adverse effects have been seen in animal studies c̄ no such effects in well controlled human trials.

Eg : Paracetamol, Ranitidine

Category C : Drugs for which there are no studies either animal or human or drugs in which there are advese fetal effects in animal studies but no such data exists in human trial

Eg: Chloroquine, acyclovir, zidovudine

Category D : Drugs which have proven risks but their use is essential & benefits outweighs the risks

Eg : Phenytoin, warfarin, propylthiouracil.

Category X : Drugs which are clearly teratogenic, & risk outweighs the benefits → Hence should be avoided.

EPILEPSY IN PREGNANCY

- Effect of Pregnancy on Epilepsy : 50% unchanged, 30% Increase, 20% Decrease
- Epilepsy is the most common neurological disorder encountered in pregnancy
- The most common cause for Epilepsy in Pregnant woman is IDIOPATHIC and

  D/D's of Convulsions in pregnancy Eclampsia

  a. Cerebral vein Thrombosis
  b. Cerebral Infarction

- HYPOGLYCEMIA / HYPONATREMIA / HYPOCALCEMIA
- Risk of Congenital Anomalies is about 4% in EPILEPTIC Patients and there is
- 4-5% Risk of Epilepsy in the child if parents are affected.
- All Anticonvulsant drugs are associated with Congenital Anomalies (6% – one drug , 15% - Two or more drugs)
- LEVETIRACETAM IS SAFEST DRUG IN PREGNANCY
- NO CONTRAINDICATION FOR BREASTFEEDING
- All women on anticonvulsants should take folic acid : 4 mg / day for 12 wks preconception and throughout pregnancy.
- PRENATAL SCREENING : MSAFP + level II USG (watch for neural tube defects)
- THERAPEUTIC DRUG MONITORING :
  - Vitamin K 10 mg / day orally from 36 wks onward to prevent hemorrhagic disease of Newborn
  - Higher dose of Estrogen required in OCPs if patient is on PHENYTOIN,

    PHENOBARBITONE and CARBAMAZEPINE.

PULMONARY DISORDERS

PNEUMONIA

1. Many bacteria that cause COMMUNITY - ACQUIRED PNEUMONIA, such as

   STREPTOCOCCUS PNEUMONIA, are part of the normal resident flora.

2. A number of factors can upset the symbiotic relationship between colonizing bacteria and mucosal phagocytic defenses. Examples include acquisition of a virulent and invasive strain or bacterial infection following a viral infection.
3. Cigarette smoking and chronic bronchitis favour colonization with S.pneumoniae, Hemophilus influenzae and Legionella
4. Other Risk factors include ASTHMA, BINGE DRINKING, SMOKING and HIV INFECTION.

FACTORS THAT INCREASE THE RISK OF DEATH or COMPLICATIONS WITH COMMUNITY ACQUIRED PNEUMONIA :

1. CO-EXISTING CHRONIC CONDITIONS
2. CLINICAL FINDINGS : Respiratory Rate ≥ 30 min, HYPOTENSION , Pulse ≥ 125 bpm.
   HYPOTHERMIA (<35°c) , TEMPERATURE >40°c,
   ALTERED MENTAL STATUS and EXTRAPULMONARY Disease
3. LABORATORY FINDINGS : LEUKOPENIA ($< 4000/\mu L$) or
   LEUCOCYTOSIS ($> 30.000/\mu L$)

- $pO_2 \leq 60$ mm Hg or CO_2 Retention while breathing room air,
- Elevated Serum Creatinine
- ANEMIA
- EVIDENCE of Sepsis or Organ dysfunction such as ACIDOSIS or COAGULOPATHY

4. RADIOLOGICAL FINDINGS : More than one-lobe involvement, cavitation or pleural effusion

a. ANTI-MICROBIAL Treatment is EMPIRICAL. Because the majority of adult pneumonias are caused by PNEUMOCOCCI, MYCOPLASMA or CHLAMYDIA; therapy with ERYTHROMYCIN or one of its newer analogs is given.

b. In whom Staphylococcal or Hemophilus pneumonia is suspected, CEFOTAXIME or CEFTRIAXONE is given in addition to ERYTHROMYCIN Therapy.

ASTHMA

Clinical Stages of Asthma:

STAGE	pO_2	pCO_2	pH	FEV_1 (%) Predicted
Mild Respiratory alkalosis	Normal	↓	↑	65 - 80
Respiratory alkalosis	↓	↓	↑	50 - 64
Danger zone	↓	Normal	Normal	35 - 49
Respiratory acidosis	↓	↓	↓	<35

STEP THERAPY OF CHRONIC ASTHMA DURING PREGNANCY:

MILD INTERMITTENT: Inhaled β Agonists - Salbutamol, Metoproterenol, Isoproterenol, Salmetrol

MILD PERSISTENT: Inhaled β - agonists as above

Inhaled CROMOLYN - continue if taking prior to pregnancy with good response

Substitute Inhaled Corticosteroids if no response - BECLOMETHASONE, BUDESONIDE, TRIAMCINOLONE

MODERATE PERSISTANT: Inhaled β - agonists as above

Inhaled Corticosteroids as above, add oral theophylline and/or inhaled Salmeterol if inhaled medium-dose steroids inadequate.

SURGICAL COMPLICATIONS IN PREGNANCY

GENERAL SURGERY DURING PREGNANCY:

a. 2nd TRIMESTER is the safest time for surgery as the risks of teratogenesis, miscarriage and pre-term delivery are lowest.

b. Emergency surgery has to be done at any time during pregnancy

c. Operation should be done preferably by a senior surgeon, an expert anesthesist with obstetrician

d. Post operative pain relief, tocolytics and injectable progesterone are very helpful.

FIBROIDS

1. EFFECTS OF FIBROIDS ON PREGNANCY :

a. Recurrent abortions

b. Impacted posterior fibroid can lead to retroverted gravid uterus and urinary retention

c. Malpresentations

d. Pre-Term Labour

e. IUGR

f. Prolonged labor / Obstructed labour

g. Cervical dystocia

h. Abruption

i. Atonic PPH

j. Increased Risk of Obstetric Hysterectomy

2. EFFECTS OF PREGNANCY ON FIBROIDS

a. Red degeneration

b. Increase in size

c. Torsion

d. Dysfunctional labour

e. PPH

f. APH

g. Placenta Previa

h. Malpresentations

RED DEGENERATION (also known as CARNEOUS DEGENERATION)

i. Occurs because fibroid overgrows its blood supply (micro necrothrombosis)

ii. Most commonly occurs in 2nd TM of pregnancy followed by in the puerperium

iii. Cut section : RAW BEEFY Appearance, Fishy odour

iv. Patients presents with acute abdomen, vomiting, fever and leukocytosis

v. D/D : Acute Appendicitis, Pyelonephritis and Abruption.

MANAGEMENT

a. Always conservative management (never surgery)

b. Hospitalization

c. Bed rest

d. Analgesics

e. i.v fluids

f. i.v Antibiotics (SOS)

PROM - Premature Rupture Of Memb.

• Diagnosis by

1) P/S examination : If we see clear fluid coming out from the OS.

• PPROM - Preterm PROM if it occurs < 37wks.

• Ⓝ Vaginal pH = 4 ; Ⓝ AF pH = 7.1

2) NITRAZENE PAPER TEST

3) FERNING : Normally in Pregnancy; if we make smear from vaginal secretion → NO FERNING

h/o Leakage of Fluid → FERNING ⊕

4) USG - OLIGOHYDRAMINOS

5) DYE TEST - Put dye in AF

• Whenever Rupture of membrane : ↑ Risk of Infections (Intra Uterine Infection)

↓

aka CHORIOAMNIONITIS → T.O.P

Management of PROM

if ≥ 34wks

↓

T.O.P + Antibiotics + Look for Infections

↳ Induction Of Labour (IOL)

if <34wks

Steroids for Lung maturation → Not given in k/c/o CHORIOAMNIONITIS

+

Routine Antibiotics

+

Look for Infections

after attaining lung maturity → T.O.P (IOL)

TREATMENT

TRUE LABOUR PAIN

• Stage of Labour

1st stage : from ONSET of TRUE LABOR pains to full cervical dilatation

2nd Stage : from full dilatation to EXPULSION of FETUS

FALSE LABOR PAIN

• Rest + Sedation

1st STAGE → LATENT PHASE : 0-5cm of Cervical dilatation [ACOG]

↘ ACTIVE PHASE : ≥6cm of Cervical dilatation [ACOG]

WHO PARTOGRAM

Latent : 0-3cm

Active : ≥ 4cm

LATENT PHASE	Ⓝ	Prolonged
PRIMI	12hrs	≥ 20hrs
MULTI	8hrs	≥ 14 hrs

Latent Phase if Prolonged
↓
Adequate REST and SEDATION
(So that she is not exhausted)
↓
Augmentation of Labour
(↑ Intensity, Frequency and Duration)
HOW?

By Giving ⟶ ARM ⟶ Ruptures
↓
Releases PGE

By Giving ⟶ DRUGS (Preferred)
- OXYTOCIN (Preferred)
- MISOPROSTOL (PGE$_1$ Analogue) ⟶ HYPERSTIMULATION OF UTERUS

Q. Pt in labour; 8am - Dilatation 1cm, 80% Effaced, Memb. ⊕. Other things Ⓝ

8pm - " 2cm, 80% " , " . What Next?

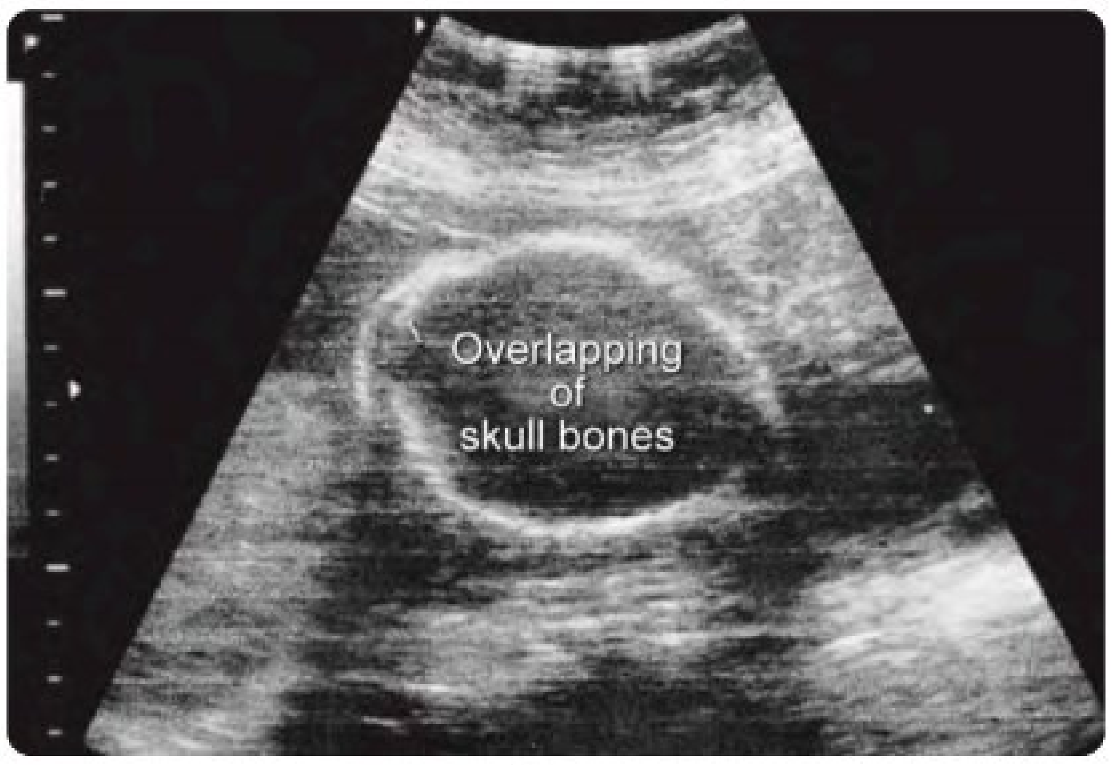
Ans: It is TRUE LABOUR, 1st Stage - LATENT. So, advice REST and SEDATION.
↓
Inj. PETHIDINE + PHENERGAN
↓
New Labour Analgesia ⟶ EPIDURAL, T_{10}, BUPIVACAINE

IUD

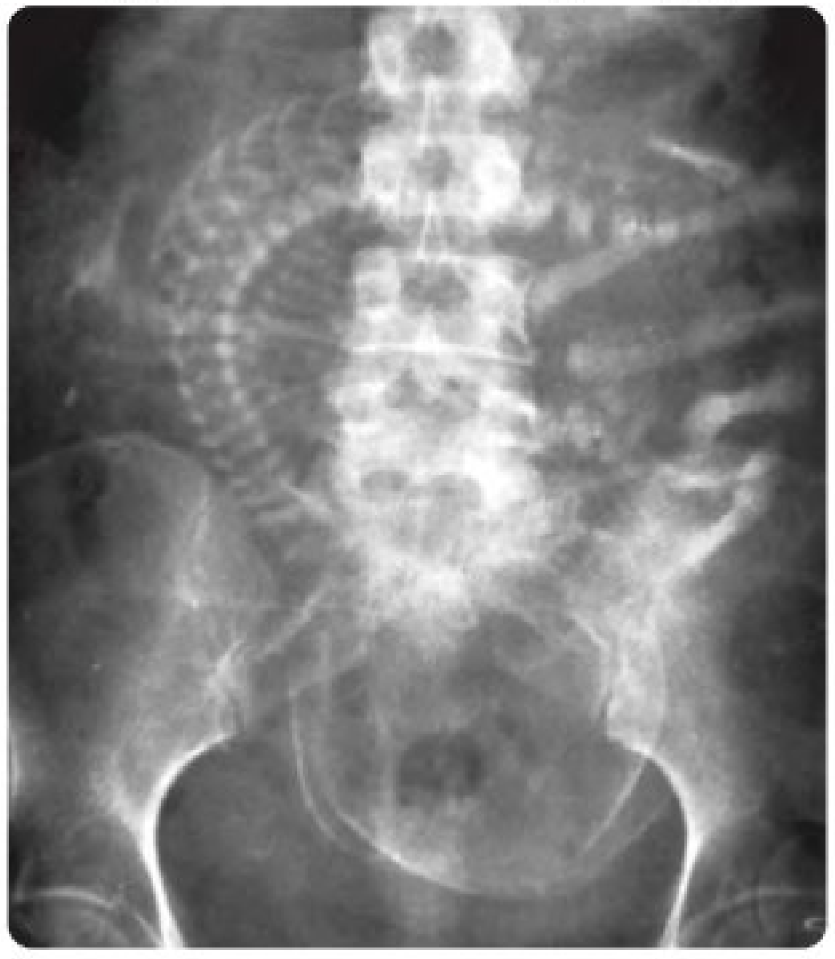
Signs of IUD on X-Ray

q 1st sign = ROBERT SIGN :
- Presence of gas in great vessels of fetus
- Becomes +ve at 12-24 wks of fetal death

2nd sign = SPALDING SIGN :
- Overlapping of cranial bones of fetus
- Becomes positive within a wks of fetal death



3rd sign : BALL Sign → Hyperflexion/Hyper extension of fetal spine



3. Neonatal Complications
- Premature
  - ↓ RDS (+ Lung maturity is delayed)
- Hypoglycemia (<40mg/dl)
- ↓ K^+
- ↓ Ca^{2+}
- ↓ Mg^{2+}
- Hyperviscosity Syndrome
  - ↓ Polycythemia + Hyper bilirubinemia

Anemia & Mental Retardation are not seen in babies of Diabetic mother

* Best test for assessing Lung maturity in fetus of diabetic mother

(OR)

overall for assessing Lung maturity in fetus

→ Phosphatidyl Gilycerol

- Present in Amniotic fluid
  ↓
  Lungs matured
- Absent in Amniotic fluid
  ↓
  Lungs not ma

Q Tocolytics C/I in Diabetic Patients

β-agonist → Ritordine
Isosuxprine
Tertabutaline
Salbutamol

Q Tocolytics of choice in Diabetic patient → Nifedipine

Q All are seen in gestational diabetes except

AIIMS May 10

a) Previous macrosomic baby
b) Obesity
[✓ c) Malformation]
d) Polyhydramnios

Q The MC congenital anomaly seen in pregnancy c̄ diabetes is

AIIMS May 03

a) Multicystic Kidney
b) Oesophageal atresia
[✓ c) Neural Tube defects]
d) Duodenal atresia

Q MC congenital malformation in diabetic pregnant woman amongst following ar

AIMPG 07

[✓ a) Cardiac defect]
b) Renal defect
c) Liver defects
d) Lung defects

Q Best test for fetal maturity in a diabetic mother is,

AIIMS June 00

a) L:S Ratio
b) Lecithin - Cephalin Ratio
c) Phosphatidyl choline
[✓ d) Phosphatidyl glycerol]

Q What should be the time of termination of pregnancy of a female with Insulin dependent diabetes

AIIMS Nov 18

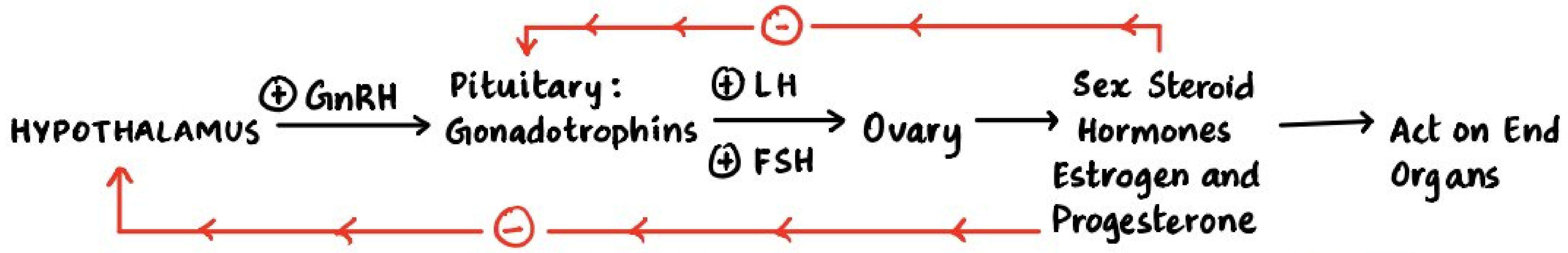
[✓ a) 40wks]
b) 38 wks
c) 37 wks
d) 34 wks

GDM (diet) : 30 wks to 40 +6days

GDM (Insulin) : 30 wks to 30 wks + 6 days

Overt diabetes : 37 wks to 37 wks + 6days

Gynaecology



⊖
HYPOTHALAMUS
⊕ GnRH
Pituitary: Gonadotrophins
⊕ LH
⊕ FSH
Ovary
Sex Steroid Hormones Estrogen and Progesterone
Act on End Organs
⊖

OVARIAN HORMONES

Potency order: $E_2 > E_1 > E_3$

I. ESTROGEN

- C_{18}, Naturally occuring

E_1 - ESTRONE - Post menopausal

E_2 - ESTRADIOL - Reproductive phase

E_3 - ESTRIOL - Pregnancy

OCP's

1. High dose - ≥ 50mg EE (No longer used)
2. Low dose - 30-35 mg EE
3. Very Low dose - ≤ 20 mcg EE
4. Lower possible dose - 10mcg EE

SYNTHETIC ESTROGENS

- Ethinyl Estradiol (EE) present in combined OCP (E+P)
- Most among all: EE > CEE > $E_2 > E_1 > E_3$
- CEE (Conjugated Equine Estrogen): dose 0.625mg

SOURCE:

1. E_1 - Post menopausal women

- from Androgens: Androstenedione $\xrightarrow[\text{tissue}]{\text{happens in Adipose}}$ E_1
- AROMATASE: Terminal Enz in formⁿ of Estrogen. (Obesity linked to cancers → E_1)

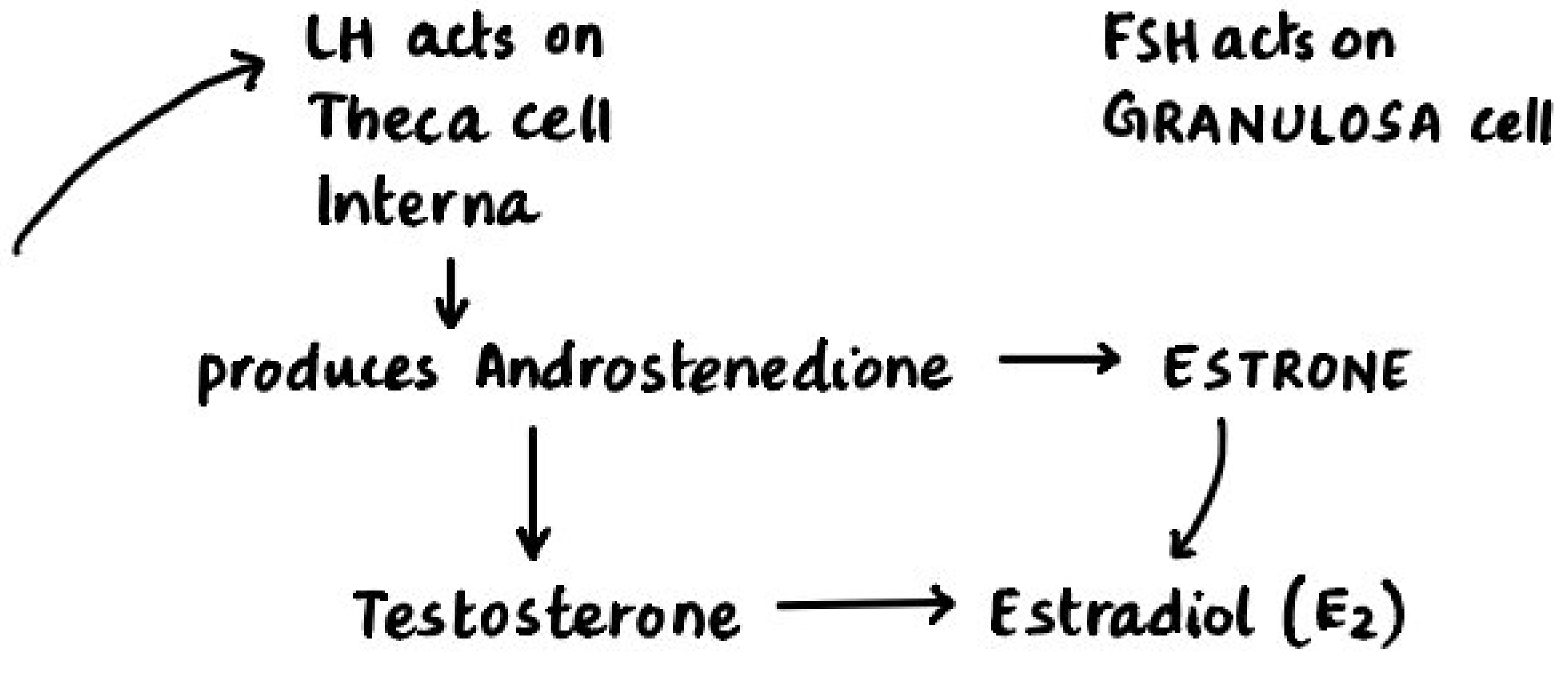
↓

Aromatase Inhibitors: · Hypoestrogenic State · given for pathologies which are Ⓔ dependent; but never 1st Line drugs - not well tolerated.

2. E_2 - Reproductive Life

- from GRANULOSA cells of follicles
- 2 cell - 2 gonadotrophin hormone theory

  2 cells - Theca cell and Granulosa cell



- 2nd source - CORPUS LUTEUM

3. E_3 - Pregnancy: Placenta, DHEAS. Placenta lacks 17 α Hydroxylase / Sulphatase

PRESENT IN BODY

- 1% Free
- Binds SEX Hormone Binding Globulin (mainly) ; also binds c̄ ALBUMIN. Not c̄ CBG
  (CBG- Cortisol Binding Globulin)
- End pdt of metabolism : GLUCURONIDES
  ↳ excreted in urine (Sulphonides)

RECEPTORS LOCATION

- INTRA-NUCLEAR SITE of ACTION: INTRA-NUCLEAR

EFFECT ON ORGANS

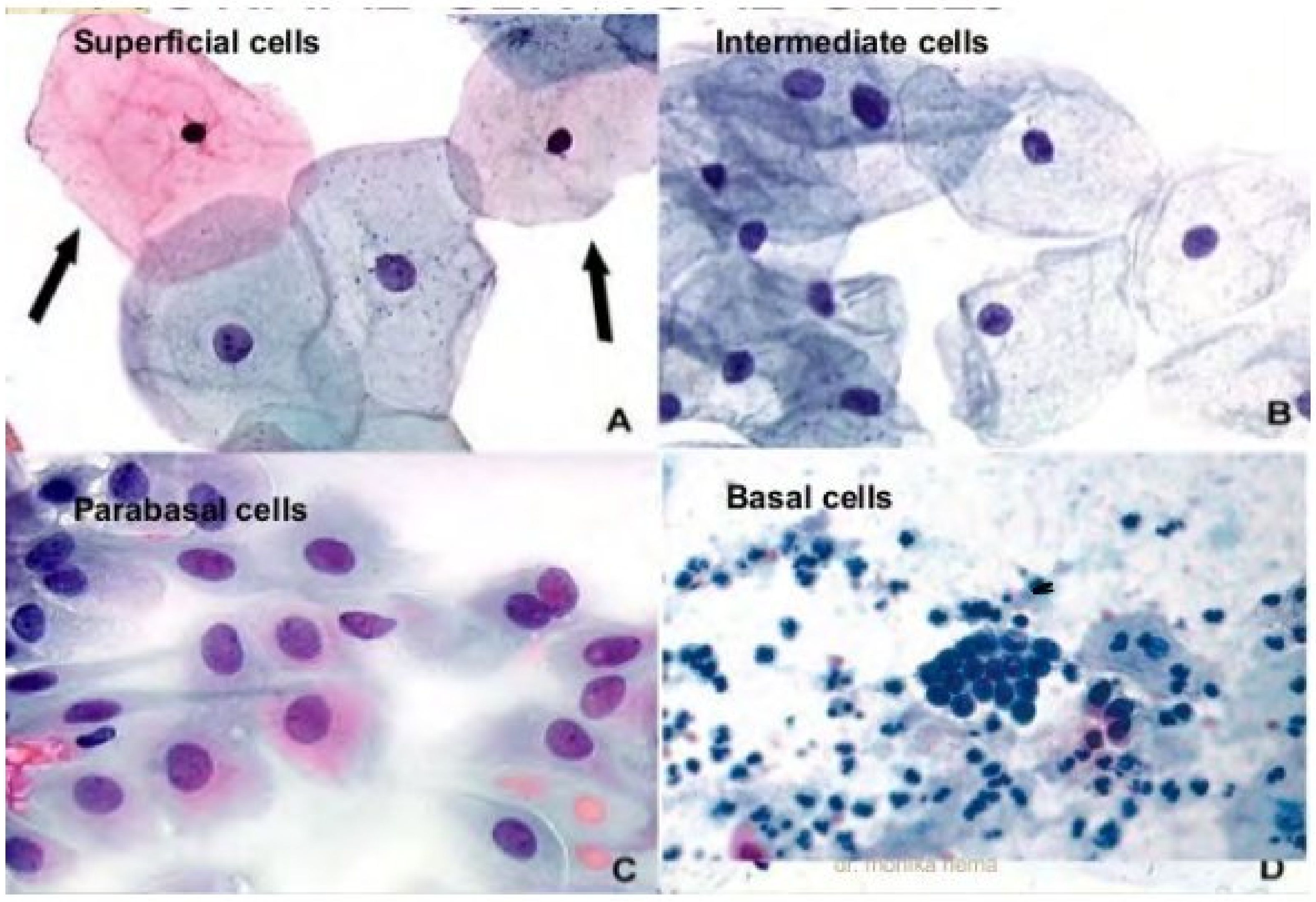
i) UTERUS : growth of smooth muscle. → FIBROIDS

ii) ENDOMETRIUM : Proliferation, on continous exposure / excess → MALIGNANCY
↳ eg: Endometrial Hyperplasia
Endometrial Cancer

mucous keeps on stretching
↑

iii) CERVICAL MUCUS :
- Copious, Thin, Watery, SPINNBARKEIT, Ferning → d/t E + ↑NaCl
- App. on D_8 of the cycle and Disappears by D_{18} (D_1 - 1st day of LMP)
- Permeable to sperms

iv) VAGINA : Vaginal smear Cytology →



Parabasal cells → IMMATURE Cell
↓
Intermediate cells
↓
Superficial cells → Mature ; Pink c̄
pyknotic small nucleus
contains SECRETIONS

- predominant Ⓔ ; predominant Superficial cells
- can be used as test of ovulation
- Ovarian tumor - ↑Pink cells - Estrogen
  ⇒ GRANULOSA CELLS
- Parabasal cells / Basal cells - s/i Post menopause

MATURATION INDEX : Parabasal : Intermediate : Superficial

Pregnancy : 0/90/10

Secretory phase 0/70/30

Post menopausal
Post delivery
Pre-pubertal
} 100/0/0 ; 90/10/0

- Estrogen causes SALT and WATER Retention
  ↳
  Cholesterol - ↑HDL, ↓LDL, ↑TG : TOTAL CHOLESTEROL - ↓↓ (CARDIOPROTECTIVE

COAGULATION PROFILE : Pro coagulant - 2,7,8,10

VTE
Stroke } Ⓔ is C/I
CAD

v) Bone : mineralization of bone

Post menopausal Osteoporosis : DOC - BISPHOSPHONATES

Precocious Puberty : Final height short d/t will cause closure of Epiphysis

S. Ca^{2+} level - decrease

vi) Breast tissue - DUCTULAR Development

2. PROGESTOGENS

- Natural Progesterone
- C_{21} Synthetic Progestins :→ Androgenic S/E

- **1st Gen :** NORETHINDRONE
- **2nd Gen :** LNG (Levonorgestrel) : lesser S/E
- **3rd Gen :** Gestodene, Desogestrel, Norgestinate
- **4th Gen :** ANTI-ANDROGENIC Action - Spironolactone derivatives
  DIENOGEST

↓ Effect on LIPID profile improves.

- OCP's Generation depends on Ⓟ component (MC) used → 3rd Gen : DESOGESTREL
  (its a derivative of Etonogestrel)

  Spironolactone derivatives : DROSIPIRENONE

SOURCE

- Corpus luteum → formed by process called LUTENIZATION OF GRANULOSA CELLS

FSH ↘ Granulosa cell ↗ Estrogen
LH ↗ Granulosa cell ↘ Progesterone
↳ Has Receptors for Both LH and FSH → Receptor appear in the Late Proliferative Phase

(LH → • Responsible for Steroidogenesis)
• Maintains Corpus luteum

- Main hormone produced by Corpus luteum is PROGESTERONE but also pd ESTROGEN; also pd INHIBIN A; RELAXIN

↖ FSH → INHIBIN-B - Source of Granulosa cell follicle

PRESENT IN BODY

- 2% free
- Mainly binds to Albumin and also c̄ CBG
- doesnot bind to SBG

- End pdt of Metabolism : PREGNANEDIOL

RECEPTOR LOCATION

- INTRACYTOPLASMIC. Site of Action: INTRANUCLEAR.

EFFECTS ON ORGANS

i) UTERUS: Indirect Role

ii) ENDOMETRIUM: Stops proliferation, secretory changes.
If continuous Progesterone to women
→ ATROPHY of Endometrium

So, Rx of Endometrial Hyperplasia – Progesterone given continuously

iii) CERVIX MUCUS:
Scanty, Thick, viscous,
NO Spinnbarkeit, NO Ferning } TACK Phenomenomenon
- mucous breaks.
↳ Impermeable to sperms

iv) FALLOPIAN TUBE: ↓ motility, ↑ secretions
So POP – R/F for ECTOPIC Pregnancy

v) VAGINA: · predominant Ⓟ, predominant INTERMEDIATE CELLS.

Progesterone causes EXCRETION of SALT and WATER
↳ ↓ HDL, ↑ LDL

- NO EFFECT on Coagulation Profile and Bones

vi) BREAST TISSUE: GLANDULAR development

CONTRACEPTION

1. BILLING's Method – Cervical mucus; Natural method

2. SYMPTOTHERMAL Method – Natural method → Based on: Cervix mucus + Basal Body Temperature
↓
under effect of Progesterone BBT will ↑ by 0.5°F

- Least Failure Rate (Most Effective) - among natural method

PERIOVULATORY Phase – Ⓔ Type – Fertile

POP's (Progesterone Only Pill)

- Hormonal methods of Contraception ⟶ Main Mech of Action: Inhibition of Ovulation

Contraceptive Methods don't act by INHIBITION OF OVULATION

1. MINIPILL – MOA: Alteration of cervical mucus

2. IUD – inhibits Fertilization and Implantation

3. CENTCHROMAN – ORMILOXIFENE (SERM) – Brand name: CHHAYA
(Non-Steroidal Contraceptive Method) MOA: Inhibition of Implantation

MIRENA - Hormonal IUD (LNG): under the effect of Progesterone - Impermeable for micro organism → ↓ Risk of PID

VAGINA: Embryology → DUAL development: upper $1/3^{rd}$ - Mullerian ducts - MESODERMAL

Vaginal mucosa - ENDODERM

Lower $2/3^{rd}$ - Urogenital sinus - ENDODERM (Sino vaginal Bulb)

- Lining Epithelium: Stratified Squamous Epithelium
  Secretions - ⊕, ACIDIC, NO Glands
  ↓
  Main Source: Cervical glands → Alkaline Secretions → LACTOBACILLUS aka DODERLEIN's Bacilli
  ↓
  Acts on GLYCOGEN → Secretions: ACIDIC Ⓝ pH: 3 - 4.5

- if Estrogen - Absent → pH > 4.5 s/i POST MENOPAUSAL WOMEN and in PRE-PUBERTAL GIRLS/during MENSES: pH 6.5 - 7.5

- if Lactobacilli - ABSENT; pH > 4.5 BACTERIAL VAGINOSIS

- In Pregnancy Ⓝ pH = 4; in Newborn girl Ⓝ pH = 5.5 - 6

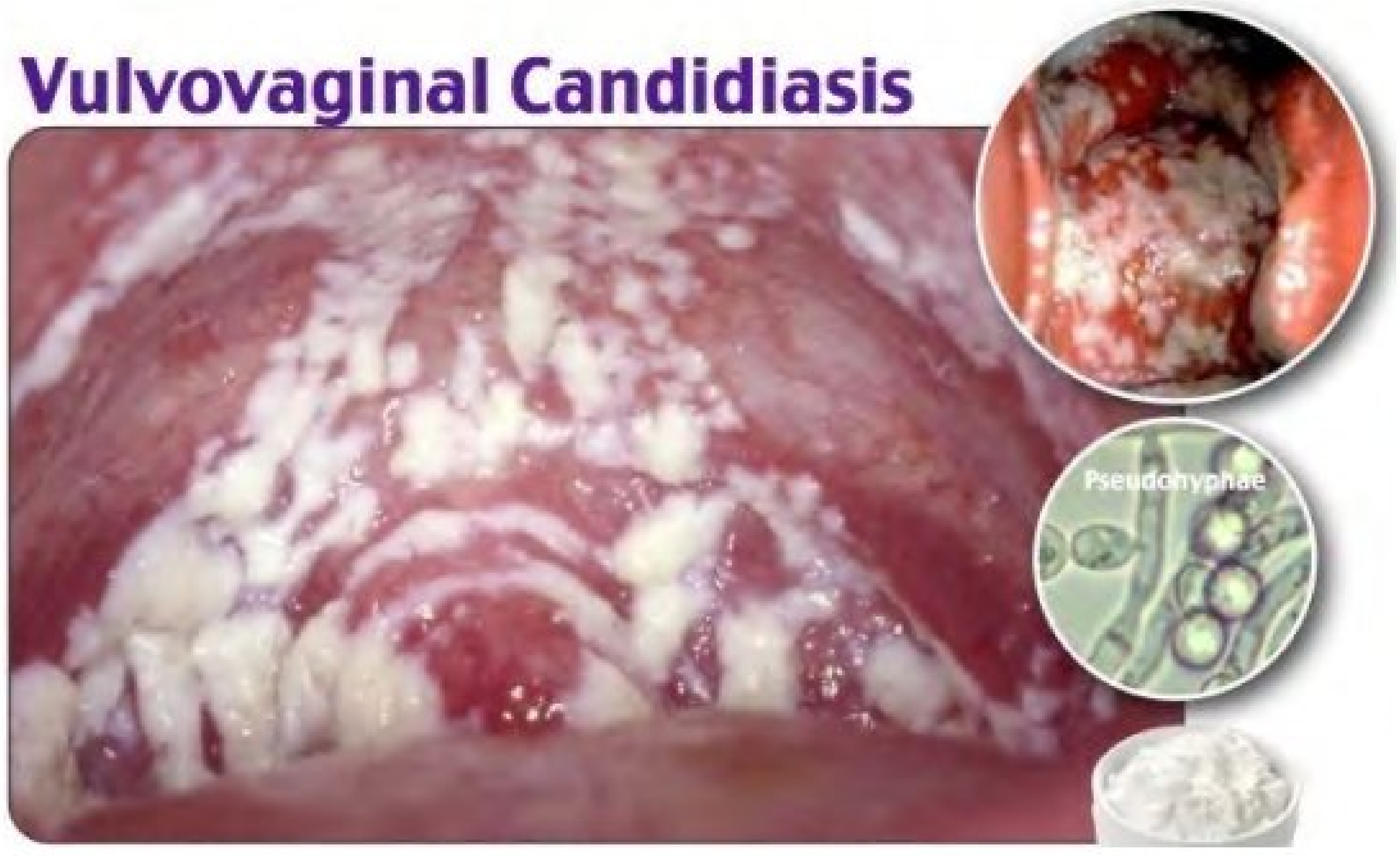
- ACIDIC Vaginal pH - protects from Infections.

If pH changes: CANDIDA present; as COMMENSA overgrows cause CANDIDIASIS ← s/i Immuno compromised DM

- VULVOVAGINAL CANDIDIASIS - not an STD - So NO NEED for Partner Rx.

CENTCHROMAN TABLETS I.P.
Non-Steroidal Oral Contraceptive Pills
8 Tablets
Chhaya
*Twice a week for first 3 months
*Once a Week after 3 months
Government of India
FREE SUPPLY, Not for Sale

Vulvovaginal Candidiasis



ESTROGEN - Will upregulate Progesterone Receptors on ENDOMETRIUM
↓
Progesterone will act only on Estrogen primed Endometrium

1st Half of Menstrual Cycle: ESTROGEN - predominant hormone

2nd Half of Menstrual Cycle: PROGESTERONE - predominant hormone

ENDOMETRIAL THICKNESS <
- if Thick - Estrogen Primed
- if Thin - Not primed

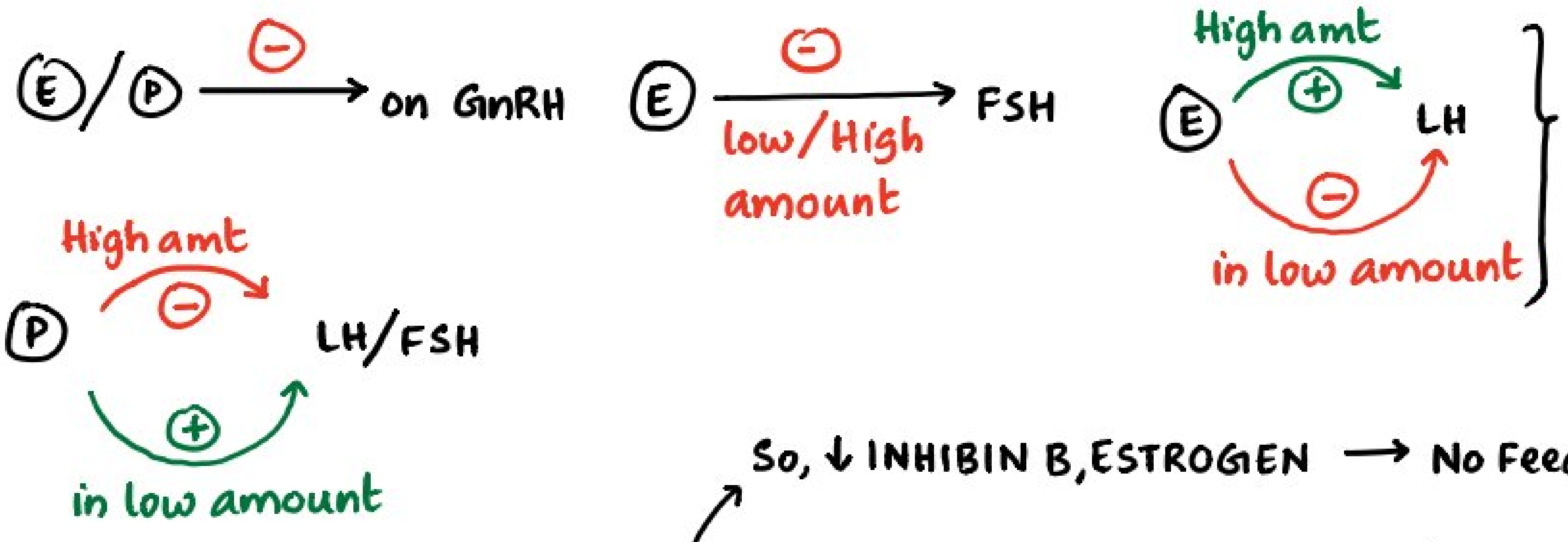
Q. 38yr old ; Bleeding P/v x 15 days ; UPT - Negative , Endometrium - Thin. What next ?

Rx: 1st give ESTROGEN : temporarily stops bleeding by proliferation → then Replace c̄ Progesterone

Q. 38 yr old women ; no bleeding for past 2 months (amenorrhoea) , UPT Negative ; Endometrium will be Thick

Rx: Give Progesterone → On withdrawal will starts bleed but not heavily

PROGESTERONE will downregulate ESTROGEN Receptors on ENDOMETRIUM → hence stops PROLIFERATION.



NEURO ENDOCRINAL Phenomena

So, ↓ INHIBIN B, ESTROGEN → No Feedback on FSH levels ↑

- Post menopausal (as age ↑, Follicles ↓)

Ovary Ⓝ $\xrightarrow[\text{Estrogen} \ominus]{\text{Inhibin B}/}$ FSH maintained at LOW levels

ANDROGENS

- **TESTOSTERONE**
- **Main Source** : 50% - Peripheral conversion of ANDROSTENEDIONE

  25% - Adrenal gland ; 25% - Ovary

OVARY → THECA EXTERNA

- DHEA S (sulphated) - only in Adrenal gland.

↳ Highest amount of which Androgen : Androstenedione > DHEA > Testosterone

- TESTOSTERONE $\xrightarrow{5\alpha \text{ Reductase}}$ DIHYDROTESTOSTERONE → Not produced by OVARY (Active form)

  ↳ Bound form in: Females - 1% 2% Free in Males

  ↓ higher affinity Binds c̄ SHBG / Albumin

- Testosterone $\xrightarrow{\ominus}$ Synthesis of SHBG. End product - KETOSTEROIDS / OXOSTEROID
- Receptor - location INTRACYTOPLASMIC
- gene for Androgen Receptor located → on long arm of 'X' Chromosome.

- **EFFECTS**

i) Pubic and Axillary hair – Androgens

ii) Control of LIBIDO (Sexual drive)

iii) Spermatogenesis : Intratesticular levels of Testosterone is HIGH

- Main Hormone required for SPERMATOGENESIS } FSH + TESTOSTERONE
  ↳ Initiation of Spermatogenesis

TBG produced by SERTOLI CELLS

TESTOSTERONE released by LEYDIG CELLS

- For follicular development – Estrogenic environment
- ANDROGEN EXCESS – follicular development arrest

PITUITARY – Gonadotropins (LH + FSH) produced from Anterior Pituitary released in Pulsatile manner ; Protein hormones ; Receptors – MEMBRANE BOUND

- FSH Receptor → GRANULOSA Cells / SERTOLI Cells
- LH Receptor → THECA Cells, GRANULOSA Cells → LATE-PROLIFERATIVE PHASE
  → LEYDIG Cells
- $t_{1/2}$: FSH - 3-4 hrs ; LH – 30 min

FSH Functions

1. Selection of COHORT of Follicles
2. Selection and growth of DOMINANT follicle
3. Final Rupture of follicle for releasing the ovum is done by FSH (Collagen breakdown)

LH Functions

1. Formation and maintenance of CORPUS LUTEUM (C.L) –

- every month C.L undergoes LUTEOLYSIS : FIXED LIFE SPAN – 14 days
- LUTEAL PHASE : begins c̲ onset of LH Surge (14 day - Constant). Eg: 40day cycle
  40 – 14 = 26
  So, ovulation occurs on 26th day.
- β-HCG : RESCUES C.L from Luteolysis

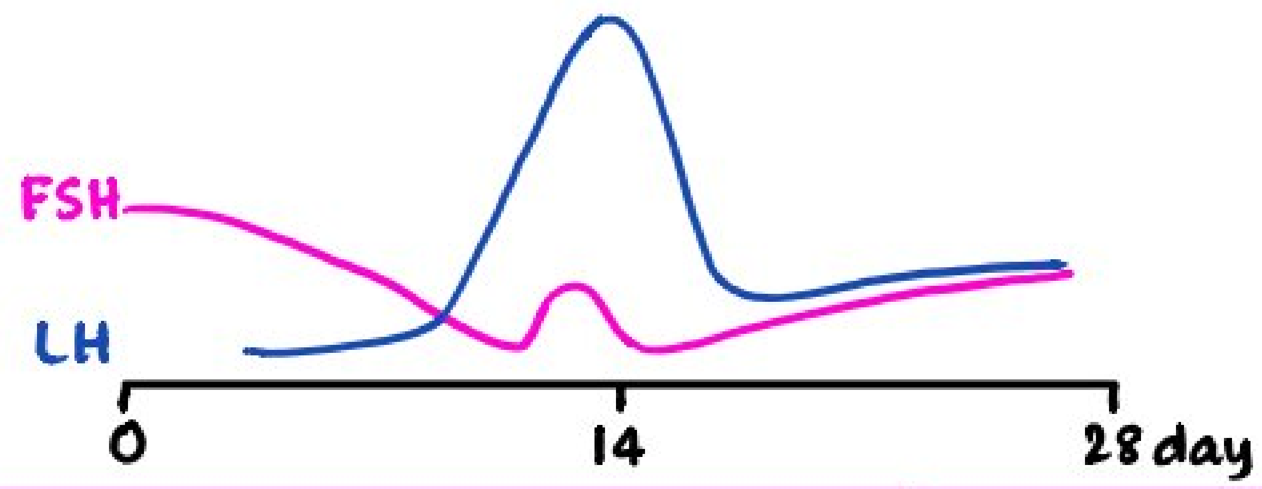
2. OVULATION

↗ Time gap: 24 - 36 hrs

- LH surge $\xrightarrow{36hrs}$ Ovulation
- LH Peak $\xrightarrow{12hrs}$ Ovulation
- For LH Surge require ESTROGEN (Minmum Req. = 200pg for atleast 48hrs)
  Highest = 400pg
- Maintenance of LH surge = Ⓟ + Ⓔ
  ↳ Low levels $\xrightarrow{\oplus}$ LH Surge

- When does Progesterone production begin? - Before Ovulation-onset of LH Surge
  ↓
  Lutenisation of Granulosa cells

- Before Ovulation : LH↑, FSH↑



3. Final growth of the follicle is done by LH

Q. When do you see peak activity of C·L? → 8 days Post ovulation (Life span = 14 days)

HYPOTHALAMUS

- GnRH - from Arcuate Nucleus.
- Neurons - derived from OLFACTORY PLACODE
  Travel along Olfactory Nr "NEURONAL MIGRATION" → ARCUATE NUCLEUS

• KALLMAN'S SYNDROME

- X Linked Recessive ; M > F
- Absent Neurol migration - no GnRH + ANOSMIA

- HP axis - dormant before PUBERTY. Pulsatile GnRH Release starts at PUBERTY.

• Neurotransmitters associated

↑ KISSPEPTIN - most important for Pulsatile release

↓ GABA - Inhibitory NT - Dormant

↓ NP-Y (Neuropeptide-Y)

↑ LEPTIN - permissive role in the onset of Puberty → Pulsatile Release of GnRH begins at Night

- LH - at night
- FSH - at both day and night

Q. Initial Menstrual cycles in adolescent girls are anovulatory. Why?

Ans: No FSH → No dominant follicle

- GnRH Pulse Frequency
  - LOW → FSH
  - HIGH → LH

- GnRH - DECAPEPTIDE Hormone $t_{1/2}$ = 3min
  ↓
  Agonist
  1. LEUPROLIDE - (MC)
  2. GOSERELIN
  3. NAFARELIN
  4. BUSRELIN

  not orally active

Pulsatile manner → Stimulate Ovary

Continuous manner → ↑LH/FSH (FLARE Response)
↓
persistently HIGH → Feedback Inhibition of GnRH

- **GnRH Antagonist** : CETROLERIX ⓂⒸ - suppress HPO axis

- Indications : same as Continuous Rx
- Newer ones can be given orally
- No FLARE Response
- Very expensive

SUPPRESSION OF HPO AXIS used in :

i) Fibroid
ii) Endometriosis
iii) Hirsuitism
iv) Precocious Puberty
v) Abnormal Uterine Bleeding
vi) Breast CA
vii) PROSTATE CA

Suppression of HPO axis
↓
Hyper Estrogenic State
↓
↑ Bone Mineral density
Add back Rx : NORETHINDRONE
↓
When to start ?
- > 6 months of GnRH Agonist (continuous)
- Start ≤ initiation of GnRH

- ovary ⟶ Endmetrial phase
- Follicular ⟶ Proliferative
- Luteal phase ⟶ Secretory phase

LUTEAL PHASE

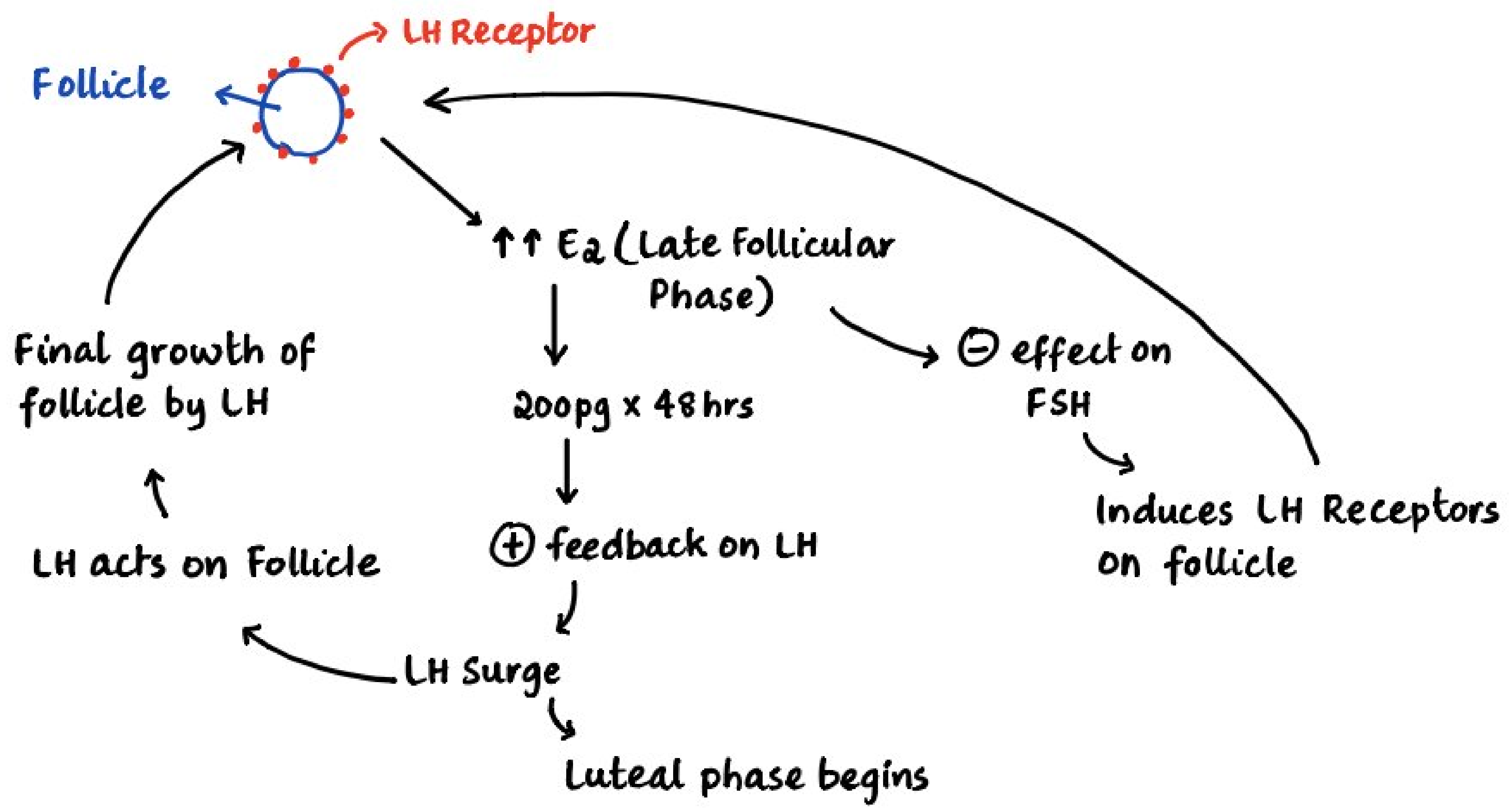
LH Surge —36 hrs→ Ovulation
LH Surge ⟶ Corpus luteum
↓
Progesterone : 3 days post ovulation - Maximum levels
Progesterone —⊖→ LH Surge

LATE LUTEAL PHASE

LH ↓ ⟶ C.L Repression ⟶ C. albicans
↓
↓ in Progresterone : aka PROGESTERONE WITHDRAWAL ⟶ Regular Monthly Bleeding " Cycles are OVULATORY"
↓
Release of PGs ($PGF_{2\alpha}$) ⟶ Myometrium (1° Dysmenorrhoea)
(vasoconstrictor)
↑
Endometrium
↓
Necrosis
↓
Shed off
↓
MENSES

ANOVULATORY CYCLES - IRREGULAR - PAINLESS

- ↓ Progesterone, ↓ Estrogen } ⟶ ? GnRH level begins to increase : Marks Onset of Follicular phase.
↓
Low frequency Release of FSH
↓
Release INHIBIN-B and E_2 —⊖→ FSH
- Keeps FSH under check so that one ≤ maximum Receptor will continue to become DOMINANT Follicle



NORMAL MENSTRUAL CYCLE

- Normal Length : 21-35 days [FIGO : 24-38 days]. Average - 28 days
- Normal Blood loss - upto 80ml [Avg: 35-50ml]
- Normal No. of Days : 2-8 days [4.5-5 days]

ABNORMAL UTERINE BLEEDING [AUB]

1. **MENORRHAGIA :** REGULAR CYCLES, ↑ Blood loss > 80ml, > 8days
2. **HYPOMENORROEA :** Bleeding < 2days (< 20ml)
3. **POLYMENORRHOEA :** Frequent cycles (< 24days, < 21days)
4. **OLIGOMENORRHOEA :** Infrequent cycles (> 38days, > 35 days)
5. **METRORRHAGIA :** Intermenstrual Bleeding (Irregular Bleeding)

FIGO DEFINITION AUB : PALM - COEIN

- P - Polyp
- A - Adenomyosis
- L - Leiomyoma
- M - Malignancy and HYPERPLASIA
- C - Coagulopathy
- O - Ovarian causes
- E - Endometrial causes
- I - Iatrogenic
- N - Not yet classified

PUBERTY

: Appearance of 2° sexual characters (Sex steroid hormones)

- **BOYS** - Age : 11.5 yrs 1st Sign : Testicular enlargement

Precocious : < 9yrs

Delayed : 14 days

1st visible sign : Testicular enlargement ⟶ Penile enlargement ⟶ Pubarche ⟶ Peak Height Velocity

- **GIRLS** Age : 10·5 yrs   1st Sign : GROWTH SPURT (↑ Arm span)

  Precocious : < 8 yrs   1st visible sign : THELARCHE (Appearance of Breast buds corresponds to TANNER Stage - 2)

  Delayed : 13 yrs

↓

PUBARCHE (Appearance of Pubic and Axillary hair ↓ Effect of TESTOSTERONE)

↓

Menarche ←After 6 months— Peak Height Velocity (Tanner - 3. Breast / Pubic Hair)

- Precocious Menarche < 10 yrs
- Average Age 12·5 yrs   • Initial cycles → ANOVULATORY
- Unopposed Estrogen : Breakthrough bleeding
- **DOC : for Irregular Anovulatory Bleeding in Adolescent girls**

1. OCP - better compliance
2. Only Progesterone : S/E ⊕. So, poor compliance
3. Mafenamic acid + Tranexamic acid → doesnot make cycles Regular
   ↓
   - given in pts who refuse hormone Rx
   - Rx in Heavy Blood Flow
4. High dose i.v Estrogen

- (MC) cause of AUB in Adolescent girls - ANOVULATION
- 2nd (MC) cause of AUB - Coagulation disorder

PRECOCIOUS PUBERTY : (MC) cause is **IDIOPATHIC**

- premature activation of HP axis → **CENTRAL**
- Peripheral **1. GRANULOSA CELL TUMOR** **2. Congenital Adrenal Hyperplasia**

3. McCune Albright Syndrome [TRIAD] : Peripheral Precocious Puberty + Cafe au lait Spots + Polyostotic Fibrous Dysplasia

4. Long Standing HYPOTHYROIDISM : ↑ TSH ∝ $\frac{LH}{FSH}$
↓
Precocious Puberty

- Central → Brain tumor (10%) → (MC) HAMARTOMA (Tuber cinerium)

Central P.P	**Peripheral P.P**
• LH / FSH ↑	• LH / FSH ↓
• Isosexual (corresponding to genetic sex)	• can be Heterosexual

Delayed Puberty : MC in Boys ; MC cause CONSTITUTIONAL DELAY - Family h/o

Constitutional cause in girls - should be Diagnosis of Exclusion

MENOPAUSE

- follicles in ovary → Maximum Number - Intra Uterine Life - 5th month = 7 million (20wks)
  ↓
- at birth - 2 million follicles
- at puberty - 4,00,000 (about 1000 follicles undergo ATRESIA every month)
- Clinical Diagnosis : Absence of menses for 12 consecutive months (in a corresponding age group)
  > 45yrs (Avg. Age : 51yrs) (Avg. Age in India : 47yrs)
- Premature Menopause : < 40yr → Needs Confirmation
- Delayed Menopause : No menopause by 55yrs ~ Needs evaluation
- Post menopausal women : Estrogen ↓ (< 20pg) · FSH ↑ (≥ 40 IU) · LH ↑ (≥ 20 IU)
- Perimenopause (CLIMATRIC):

Transition phase

MC presentation → Vasomotor symptoms → HOT FLASHES (1-5 min lasts)
- ↑ Sweating
- Anxious

↳ coincide c̄ LH Surge

- **cause** - Estrogen withdrawal, Serotonin lvl ↓, Nor-Epinephrine ↑
- Only if moderate - Severe hot flash indication to start HRT

if uterus → present → Rx. of Hot flash : E + P (P- Protects Endometrium)
↳ E+P HRT → R/F for Breast cancer

if Estrogen is C/I (eg: CAD)
↳ Rx: SSRI (Selective Serotonin Reuptake Inhibitors) - 2nd line

SERM - Not given . ANDROGENS can be given.
↳ MC SERM S/E - Hot flushes

- can present c̄ vaginal atrophy (ATROPHIC VAGINITIS)
  - Vaginal dryness
  - Dyspareunia
  - Discharge
  - Pruritus
  - Bleeding
  - Recurrent UTI

On L/E : vagina - shiny ; Thin ; Loss of Rugosity

Rx : Topical lubricants or Moisturizers (never give as Systemic)

↓ Symptoms persists

give hormones → TOPICAL ESTROGENS (creams, Rings)

- pH (>4.5) 6.5 - 7.5

Perimenopausal period : Cycles become anovulatory (ESTROGEN)

Irregular bleeding → Breakthrough bleeding ↙

Q. 43yr old presents ≤ AUB x 15 days. UPT ⊖ve

- Unstable vitals
  - ↓ D and C > i.v Estrogen
    - controls bleeding
    - Sample available for HPE
- Vitals Stable
  - ↓ 1. High dose oral estrogens - stops bleeding ≤ in 24 hrs
  - ↓ 2. OCP's / POP's

- D and C not done in unmarried girls - affects fertility ; cause ASHERMAN Syndrome

↓

- OCP / POP

Q. No menses for 3months. UPT ⊕ . Next bleeding will be high. What to do ?

Ans: Endometrium Thick, no need for priming OCP/POP

- **UTERO OVARIAN Ligaments :** attaching Ovaries to Uterus.
- **SUSPENSORY LIGAMENT / INFUNDIBULO PELVIC Ligament** - attaching ovary to lateral pelvic

  carries Uterine artery and Uterine vein
- **MESO OVARIUM** - fold of peritoneum which attaches ovary to posterior leaf of Broad Ligament
- In Hysterectomy, if ovary is to be saved ; cut the ovarian ligament
- In Hysterectomy + BSO ; then put clamps on INFUNDIBULO PELVIC Ligament
- **Ovarian veins** → Ⓡ side $\xrightarrow{\text{drains}}$ IVC

  ↘ Ⓛ side $\xrightarrow{\text{drains}}$ Ⓛ Renal vein
- Lymphatics → Para aortic grp LN

- **REFERRED PAIN OF OVARY:** travels along Lateral Cutaneous Br. of Obturator Nr. and felt on medial aspect of Thigh.
  Ureter is posterior to Ovarian fossa

EMBRYOLOGICAL DEVELOPMENT

1. GONADS

derived from genital ridge → 5wks POG

Testes Ovary

Y Chromosome → SYR Region – Short arm of Y Chr. Distal part

- Testes will appear – 7wk
- Ovary will appear – 8wk

Ovary ↓

- Absent 'Y'
- for ovary to be functionally Ⓝ → XX

- In TURNER's Syndrome (45 XO) – ovaries appear but not functional undergo Accelerated atresia
- Differentiation of gonads into Male and Female – 7wks

2. INTERNAL GENITALIA

MALE TYPE	FEMALE TYPE
Seminal vesicle Ejaculatory ducts Epididymis Ductus deferens	Uterus Vagina Fallopian Tube
From WOLFFIAN DUCTS/ MESONEPHRIC	MULLERIAN DUCTS / PARAMESONEPHRIC DUCTS

MIS / TESTOSTERONE (Male Hormones) → MIS – disappearance of Mullerian ducts (IPSILATERAL)

- Testosterone – WD – Male Internal Genitalia
- MIS – SERTOLI Cells – 7wks
- Testosterone – LEYDIG Cells – 8wks

MIS / TESTOSTERONE (Male Hormones)
↳ If ABSENT causes • persistance of MD and Disappearance of WD

- MD – remnant in Male fetus APPENDIX OF TESTIS aka HYDATID of MORGAGNI
- Appendix of Epididymis is WOLFIAN in Origin

Remnants of Wolfian Duct

1. EPOOPHORON ⟶ Mesonephric tubule
2. PARAOOPHORON ⟶ Mesonephric tubule
3. GARTNER DUCT ⟶ Mesonephric

} All 3 are contents of BROAD LIGAMENT

- **GARTNER'S CYSTIC DUCT** - seen as cyst on Anterior wall of Vagina (cyst on POSTERO-LATERAL wall of Vagina - BARTHOLIN'S CYST
- **PARATUBAL CYST** (around FT)

- Internal genitalia differentiated into Male and Female by 10wks

EXTERNAL GENITALIA

Male	Male hormone	Female
⊕	Testosterone	⊖
	↓ 5α Reductase	
	DHT	
PENIS	Genital tubercle	CLITORIS
PENILE URETHRA	Genital fold	L.minora
SCROTUM	Genital swelling	L.majora
Bulbourethral glands (COWPER's)		Bartholins glands
Prostate		Glans of Skene (Peri-urethral)

VESTIBULE

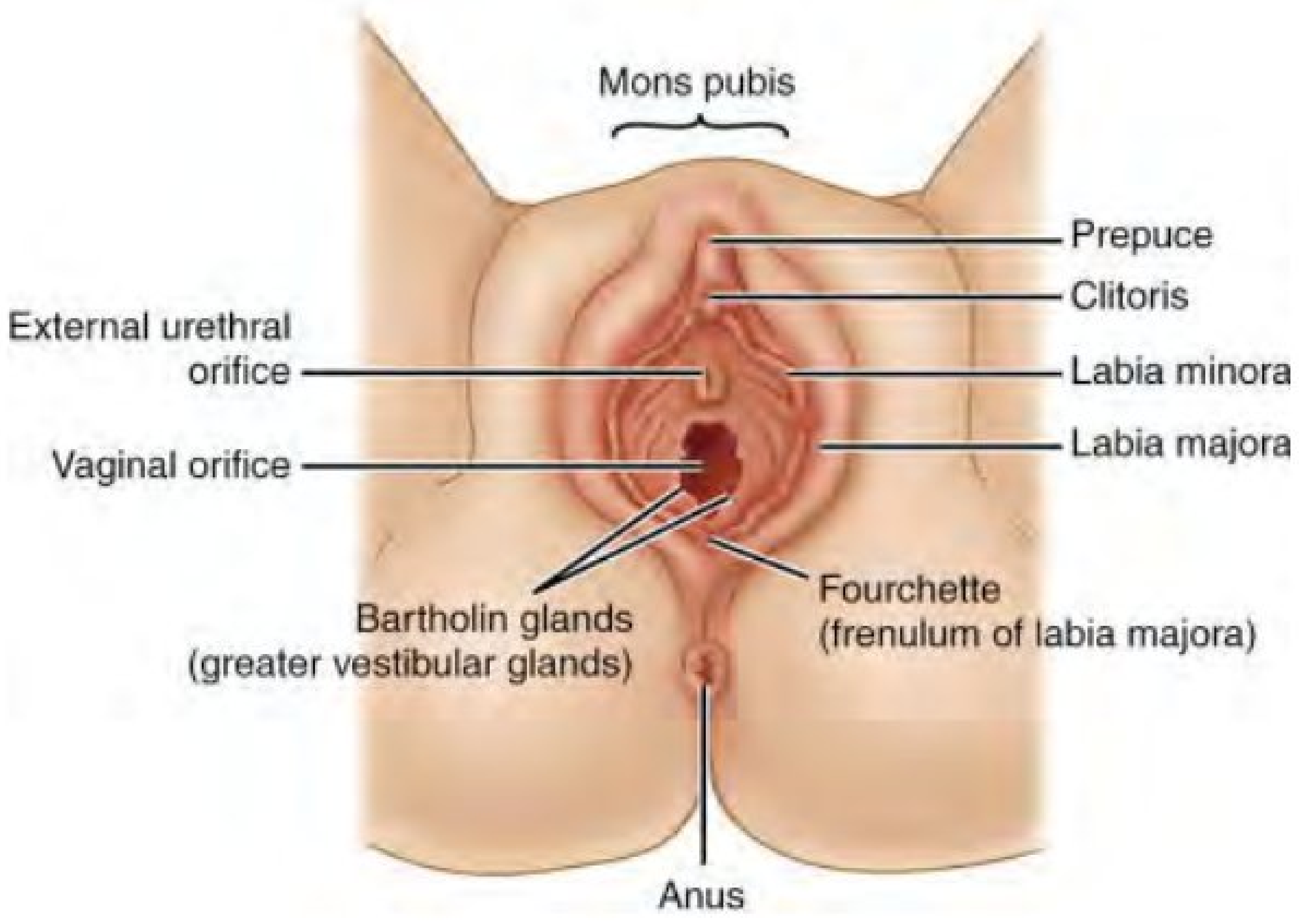
- Anterior Border - CLITORIS
- Posterior Border - FOURCHETTE
- Laterally - L.minora (2)

- 4 openings (Major) -
  - a. urethral
  - b. Vaginal
  - c. Bartholins - (2)

+

2 opening of Glans of Skene.

Total = 6 opening


Mons pubis
Prepuce
Clitoris
External urethral orifice
Labia minora
Labia majora
Vaginal orifice
Fourchette (frenulum of labia majora)
Bartholin glands (greater vestibular glands)
Anus

- Differentiation of Genitalia into Male and Female - 12wks
- Sex of fetus decided by External Genitalia
- Female hormones acts at puberty → causes → growth of the structure
- NO ESTROGEN - uterus may be present but small hypoplastic (infantile)
- Every fetus until MALE hormones act female

MALE HERMAPHRODITE (MPH)

- 46 XY , Ext. genotype - Female like , but Internal genitalia - MALE like
- 5 α Reductase deficiency
- (MC) cause – AIS (Androgen Insensitivity Syndrome)

PSEUDOHERMAPHRODITE FEMALE

- 46 XY , Man like Ext. genitalia → CLITOROMEGALY
  ↳ Fusion of L.majora
- (MC) cause Congenital Adrenal Hyperplasia
- (MC) Enzyme deficient → 21 Hydroxylase - causes AbⓃ genitalia development Only in Female fetus
- Screening Test : 17 Hydroxy progesterone

AMENORRHOEA

- **PRIMARY:** Absence of menses by 13yrs of age in the absence of 2° sexual characters (THELARCHE)

  (or)

  Absence of menses by 15yrs of age in the presence of 2° sexual characters (THELARCHE)

  - (MC) cause of 1° Amenorrhoea : Gonadal dysgenesis → TURNER's
    → Pure ; Mixed

- **SECONDARY:** Absence of menses for 90 days (3 consecutive) in a previously menstruating woman
  - irregular cycles = 6 months
  - (MC) cause - Pregnancy ; Pathological cause - PCOS

- **Investigation:** USG

if Uterus ⊕
i) Gonadal Dysgenesis
ii) Kallman's Syndrome
iii) Imperforate Hymen
iv) Constitutional

if uterus ⊖
i) Mullerian agenesis
ii) AIS

	TURNER'S SYNDROME	KALLMAN'S SYNDROME	IMPERFORATE HYMEN
1. Breast development	⊖	⊖	⊕
2. USG Uterus Ovaries	 ⊕ Hypoplastic ⊖ Fibrotic Streak	 ⊕ ⊕	 ⊕ ⊕
3. FSH / LH	Hyper gondotrophic Hypogonadism	Hypogonadotrophic hypogonadism	Ⓝ
4. Pubic and Axillary hairs	⊕ Sparse	⊕ Sparse	Ⓝ
5. Most Characteristic	Short Stature 45 XO - absence of SHOX gene which is required for growth of long bones	Ⓝ Anosmia (characteristics)	Ⓝ L/E : Bluish Bulging of membrane (characteristic)
6. Confirmation of Diagnostic (Karyotype)	45 XO	46 XX	46 XX
7. Management	HRT (E + P)	GnRH - pulsatile manner	Incision on the hymen - cruciate incision
Infertility	Donor Ovum c̄ IVF	GnRH → Ovary → Ovum	-

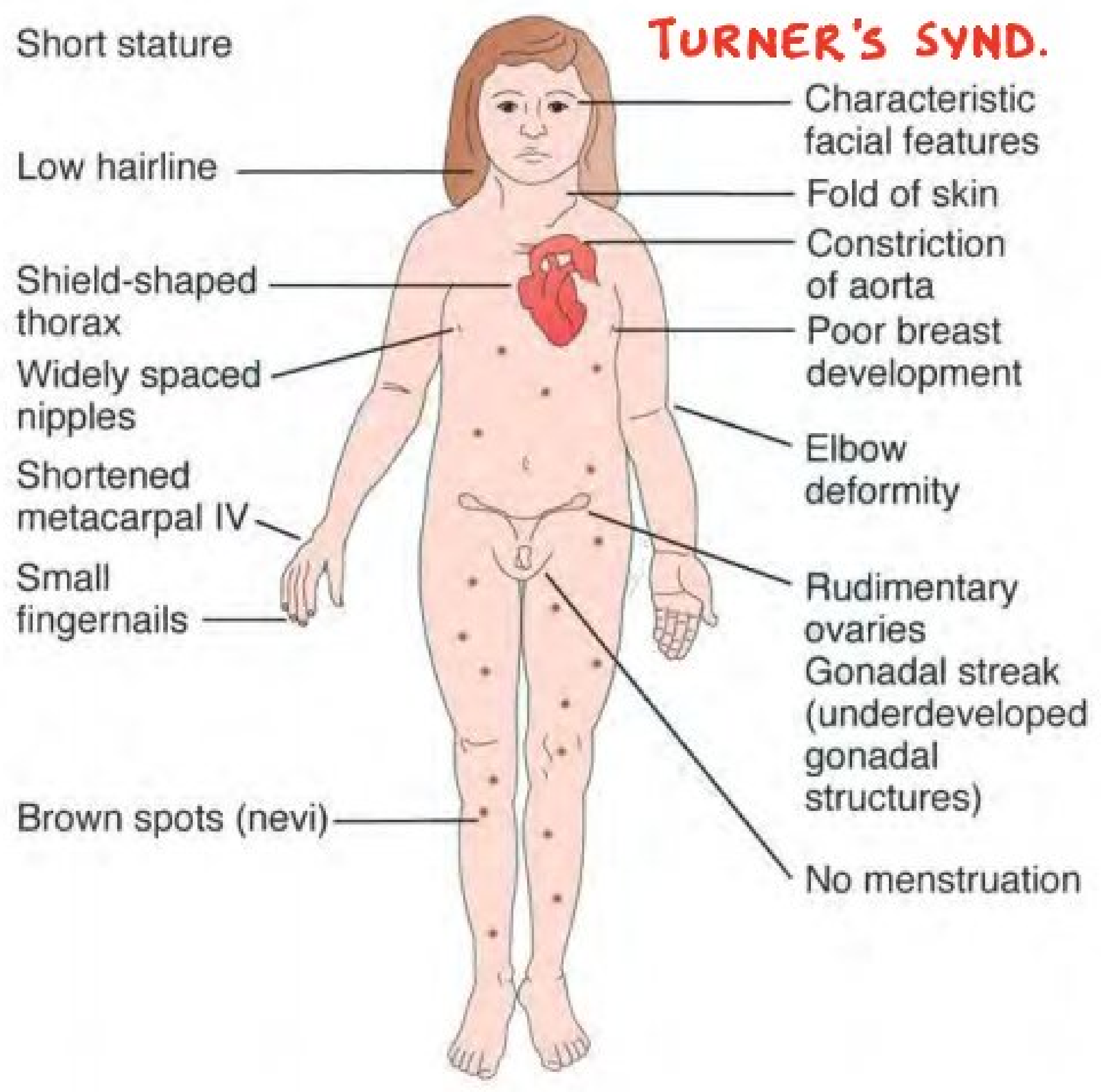
TURNER'S SYNDROME : (MC) CVS anomaly : BICUSPID AORTIC VALVE

- Short webbed neck
- Shield Shaped Chest, Nipples widely spaced
- Cubitus valgus
- IQ - Ⓝ
- Life span - Slightly Short
- Short 4th Metacarpal
- Autoimmune conditions - HASHIMOTO'S THYROIDITIS, DM.

“KLINEFELTER's (MC) Sex Chromosome AbⓃ”

KALLMAN'S SYNDROME

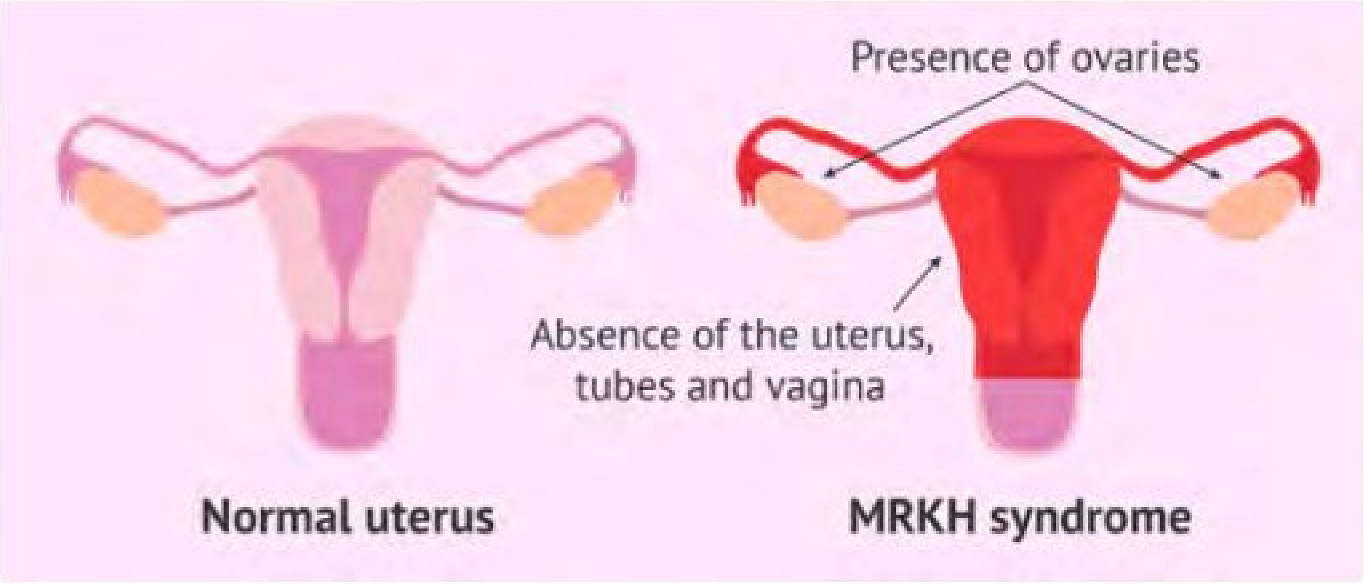
- XLR ; mutation in KAL-1 gene
- Sometimes presents as emergency :
  Blood collected in i) uterus - HEMATOMETRA P/A
  ii) Vagina - HEMATCOLPOS P/v
- can cause UTI
- can present $\bar{c}$ A/c URINARY RETENTION


TURNER'S SYND.

PURE GONADAL DYSGENESIS	MIXED GONADAL DYSGENESIS
Ⓝ Karyotype	Mosaic pattern (MOSAICISM)
46 XX 46 XY - SWYER's Synd. ↳ Mutation in SRY of 'Y' Chr.	45 XO / 46 XY → Testes Abnormal ↓ streak gonad
NO TESTES ; Streak gonads	Structure from Both ducts → Mullerian duct → Wolfian duct
No Male hormone Female phenotype	
Uterus ⊕, HYPOPLASTIC	AMBIGUOUS External genitalia
Tall Turners	Rx: HRT
height } different Karyotype }	
Rx: HRT	

- Female Phenotype - Genotype (presence of Y) ⟶ ↑ Chances of Malignancy
- Gonadectomy : done as soon as Diagnosis made. But not done in TURNER's Synd.
  ↳ done in PURE and MIXED GD
- Ⓜⓒ Tumor in GD ⟶ Gonadoblastoma (Both BENIGN and MALIGNANT)
- Ⓜⓒ Malignancy in GD ⟶ DYSGERMINOMA

PRIMARY AMENORRHOEA
USG UTERUS - ABSENT

MULLERIAN AGENESIS	ANDROGEN INSENSITIVITY SYNDROME
- ROKINTASKY Syndrome	- Testicular Feminization Syndrome
- MRKH - Type 1 (Mayer Rokitansky Kustner Hauser)	- Androgen Receptor is completely insensitive to androgens
- MRKH - Type 2 TRIAD MA + Skeletal anomalies + Renal anomalies	- XLR (Androgen Receptor on the long arm of X Chromosome)
	- ⊖ (MIS) have no uterus
- On USG : Uterus ⊖	- Testes (undescended) 46 xy
Ovary Ⓝ 46 xx	- ⊕ (Testosterone present in periphery converted to Estrogen)
Breast Ⓝ	
Pubic and Axillary hair - Ⓝ	- ⊖ Absent / Sparse vagina small blind pouch.
External genitalia Ⓝ	- female like (Hypoplastic Clitoris) (Hypoplastic Labia)
No Gonadectomy	
	- Inguinal hernia
- FSH / LH - Ⓝ	- Gonadectomy - after puberty (for sexual development to complete)
- S. Testosterone Ⓝ	
	∴ AIS = Pseudohermaphrodite ↳ (MC) cause ↗
 Presence of ovaries Absence of the uterus, tubes and vagina Normal uterus MRKH syndrome	- Testes → INHIBIN ⊕→ FSH ⇝ Ⓝ LH ⇝ ↑ Testosterone ↗ No FBI Receptor Insensitivity
	- ↑ Male level hormone
- Diagnosis Confirmation by Karyotyping	
- Vaginoplasty	- Vaginoplasty
- IVF & Surrogacy	- Worst reproductive outcome / adoption

PARTIAL AIS [REFENSTEIN SYNDROME]

- Clitoromegaly (clitoral length >1cm)
- If more Receptors are sensitive ⟶ Male phenotype maybe (possible)
- any AUB → R/o Pregnancy
- 2° Amenorrhoea → r/o Pregnancy : HCG → if not pregnant → check hormones TSH, PRL, FSH

TSH → Hypothyroidism, Hyperthyroidism : TSH $\xrightarrow{\oplus}$ TRH $\xrightarrow{\oplus}$ PRL

PROLACTIN → HYPERPROLACTINOMA : (MC) cause Pituitary adenoma (micro <10mm)

(MC) presentation : Amenorrhoea and Infertility

DOC : CABERGOLINE (Dopamine agonist)

FSH (Ⓝ = <10 IU) ⟶ if ↑ defect in ovary

↘ Ⓝ/↓ Pregnancy : 1-3 IU

1st case : PREMATURE OVARIAN FAILURE / PRIMARY OVARIAN INSUFFICIENCY

- FSH ≥ 40IU - Dx of Ovarian failure FSH ≥ 20 IU S/o
- FSH ≥ 40IU on 2 occassions 1 month ≤ ↓ in S.ESTROGEN lvl

PROGESTERONE CHALLENGE TEST

- Medroxy Progesterone Acetate (MPA) 20 × 5 days ⟶ On withdrawal ⟶ No Bleeding ↙ PCT ⊖

Management :

- HRT (E+P) upto the age of natural menopause
- Infertility ⟶ IVF ≤ Donor ovum

* Premature Ovarian Failure - Amenorrhoea + other features of menopause

2nd case : if FSH Ⓝ/↓ , $S \cdot E_2$ - Ⓝ , PCT ⊕ , Progesterone ↓ , Anovulation — PCOS

3rd case : if FSH Ⓝ/↓ , $S \cdot E_2$ - Ⓝ , PCT ⊖ - Probably d/t ASHERMAN's Synd - Intra uterine adhesions

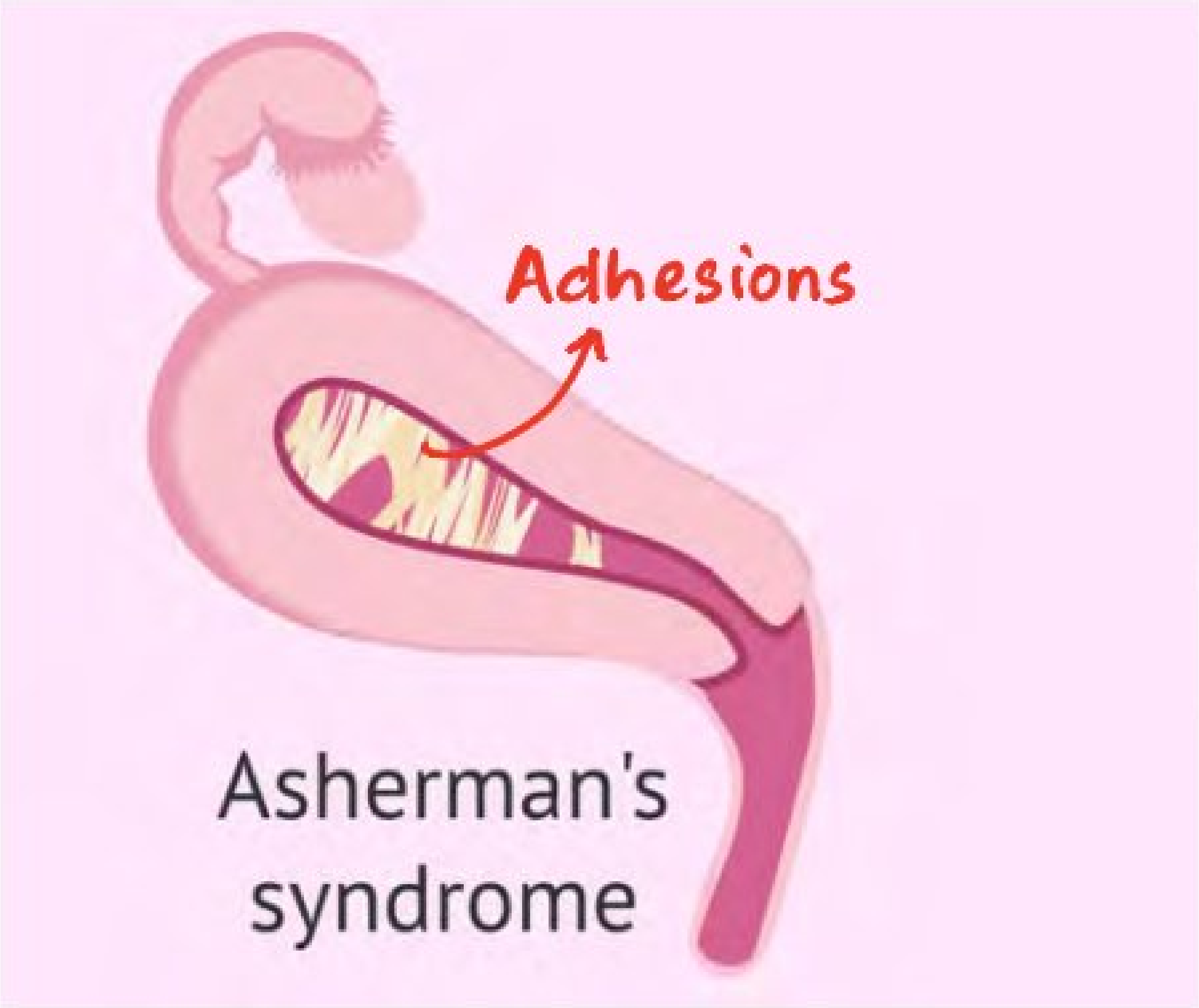
4th case : if FSH Ⓝ/↓ , $S \cdot E_2$ - ↓ , PCT ⊖ , E+P ⊕ve → Probably Central cause (FSH lvl - low)

Invgestigation : MRI - Tumor / Trauma / Infection / Infarction

- if MRI - Ⓝ , then FUNCTIONAL HYPOTHALAMIC AMENORRHOEA (Diagnosis of Exclusion)
    - Stress
    - Excercise Induced
    - Anorexia Nervosa
    - c/c Malnutrition

ASHERMAN's SYNDROME

- Intrauterine adhesions (Synechiae) → (MC) Cause Vigorous curettage
  ↳ destroy the basal layer of Endometrium (Raw surface)
- Highest Risk : Curettage in PPH ; esp. Tuberculosis
- (MC) Presentation : Menstrual irregularity → Infertility > Amenorrhoea > Hypomenorrhoea
- On HSG (Hysterosalpingography) - Screening Test
    - Multiple small irregular filling defects → s/o Ashermans
- **IOC - HYSTEROSCOPY** [Diagnostic + Therapeutic]
  ↳ Adhesiolysis
- If there is Recurrence - Intra Uterine Pediatric Foley's + High dose Estrogen x 1 month



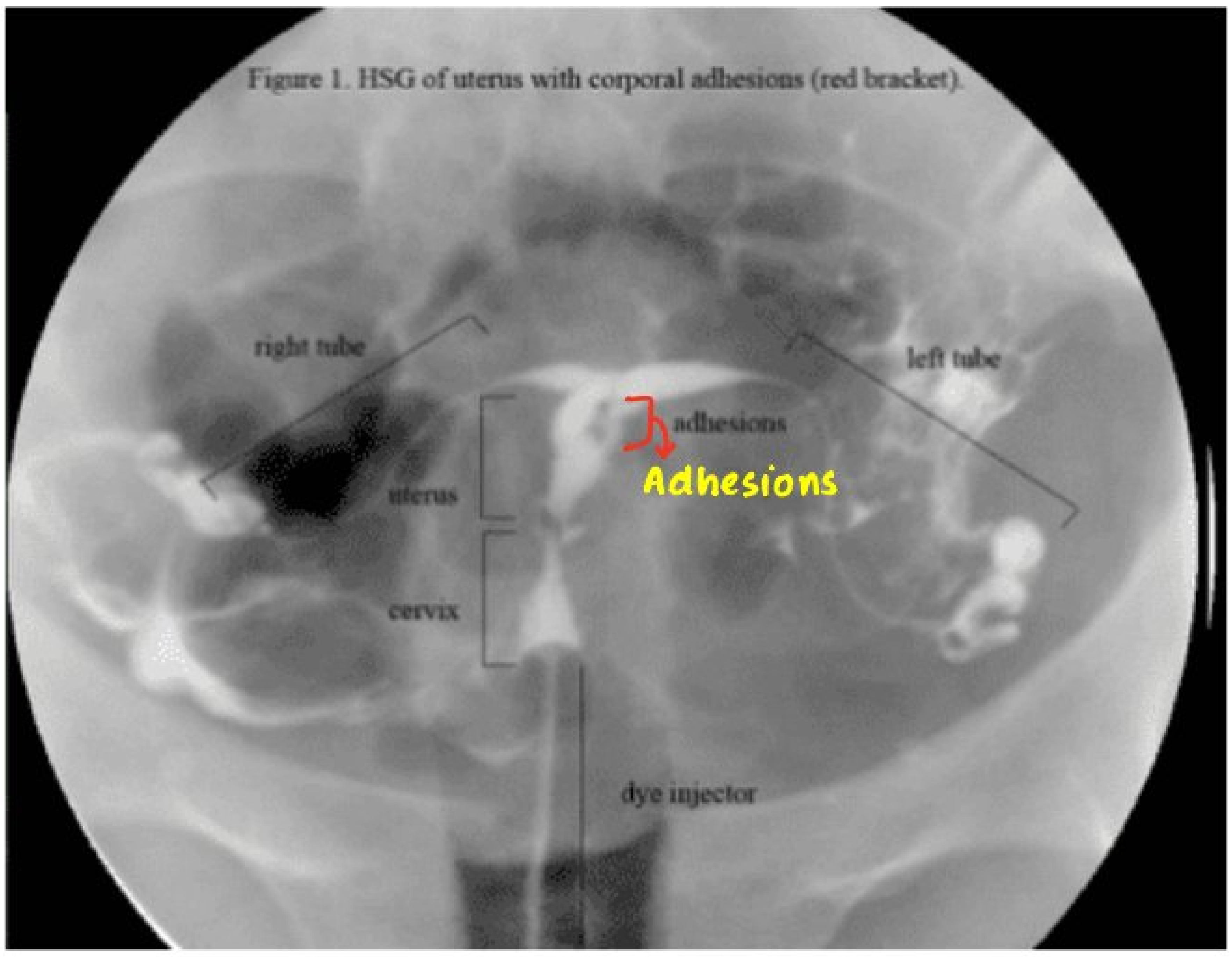


Figure 1. HSG of uterus with corporal adhesions (red bracket).

Intrauterine Adhesions on a HSG

PCOS - STEIN LEVINTHAL SYNDROME

- Ovary producing excess androgens
- Arrest of follicular growth → No dominant follicle ↳ NO OVULATION

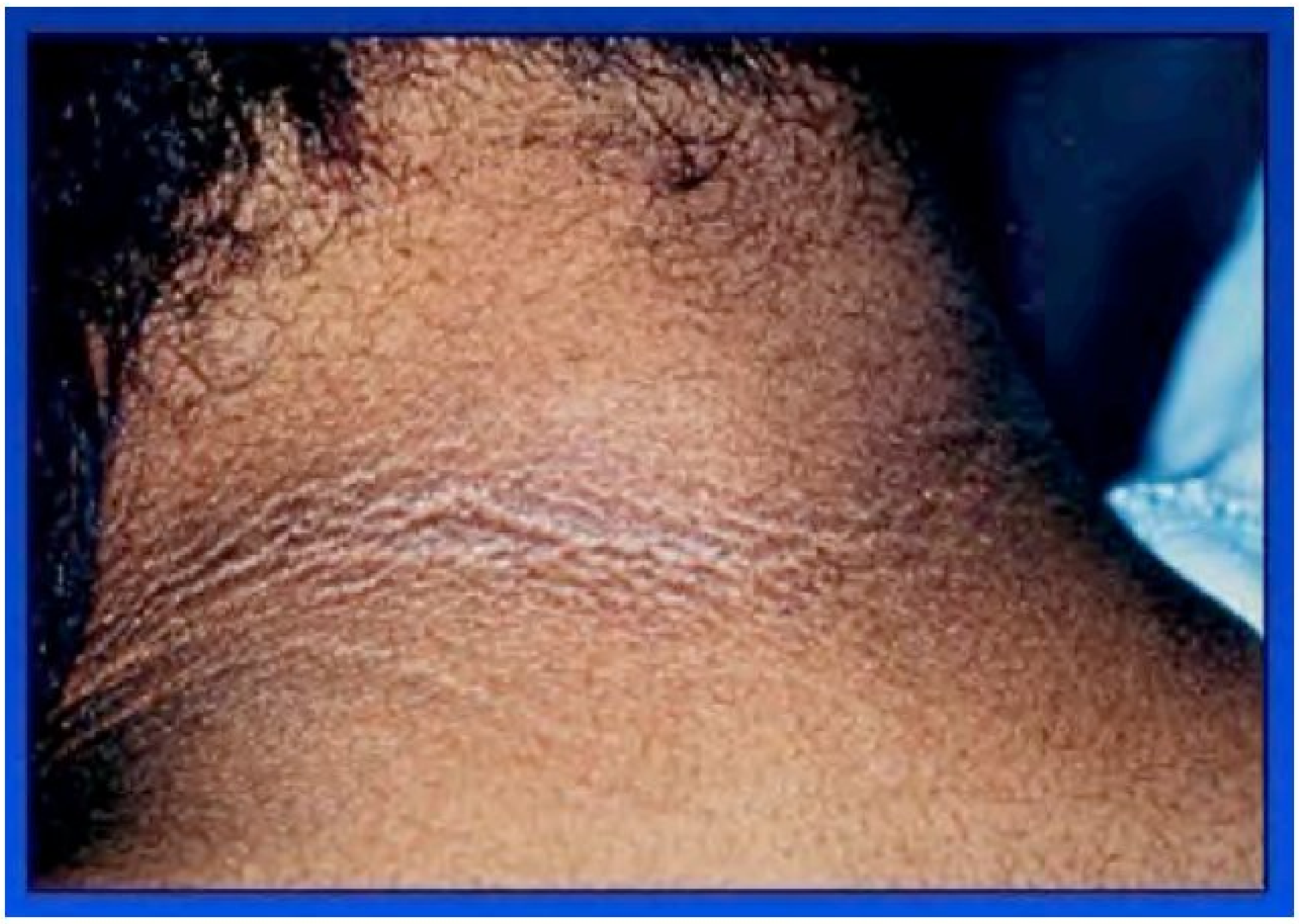
↑ Androgen in circulation
↓ Obese
E_1 (persistently ↑ LH)
LH/FSH
→ NO OVULATION

- upto 75% - also have Insulin Resistance (IR)
  Hyperinsulinemia further stimulate PCOS $\xrightarrow{\oplus}$ Dyslipidemia
- upto 50% - Obesity

→ Metabolic Syndrome (Syndrome X)

HAIR-AN SYNDROME ⊕ in PCOS

Hyperandrogenemus → ACANTHOSIS NIGRICANS - Cutaneous markers of IR



- Dark velvetty areas
- s/i Nape of neck / axilla / groin / under the breast tissue

ROTTERDAM'S CRITERIA

- if 2 of the 3 following are met, the we will make PROVISIONAL diagnosis of PCOS.

1. Amenorrhoea and / or Oligomenorrhoea

→ Anovulation (Breakthrough bleeding)

MENORRHAGIA can be a presentation but not a diagnostic criteria → Obese

- Thin girls → Rx: Withdrawal bleed by giving Progesterone

2. Hyperandrogenemia and / or Hyperandrogenism

→ Bloods levels of Androgens ↑

S. Testosterone ↑ (Mild to Moderate ↑) → in Females
→ Ⓝ upto 70 mg/dl
→ in PCOS - 150ng/dl (< 200ng/dL)

- if > 200ng - severe elevation → not s/i PCOS ; s/i Androgen producing tumors
- main Source of excess Androgen - OVARY
- Sometimes Adrenal gland ↑ Androgen secretion - DHEAS ↑ (can be a finding)

HYPERANDROGENISM – Clinical presentation

HIRSUITISM

S/i PCOS

1. Appearance of Terminal hair in male pattern distribution
2. ACNE Resistant to usual Rx → SCARS
3. ALOPECIA (Less common)

FERRIMAN GALLWAY SCORING ≥ 8 – Hirsuitism.

VIRILIZATION

S/i Tumor

1. Clitoromegaly
2. ↑ Muscle mass
3. Hoarseness of voice
4. Male pattern Baldness

3. USG CRITERIA OF ROTTERDAMS

- if i) ≥ 12 follicles which are < 10 mm in size

  ii) Ovarian volume ≥ 10cc

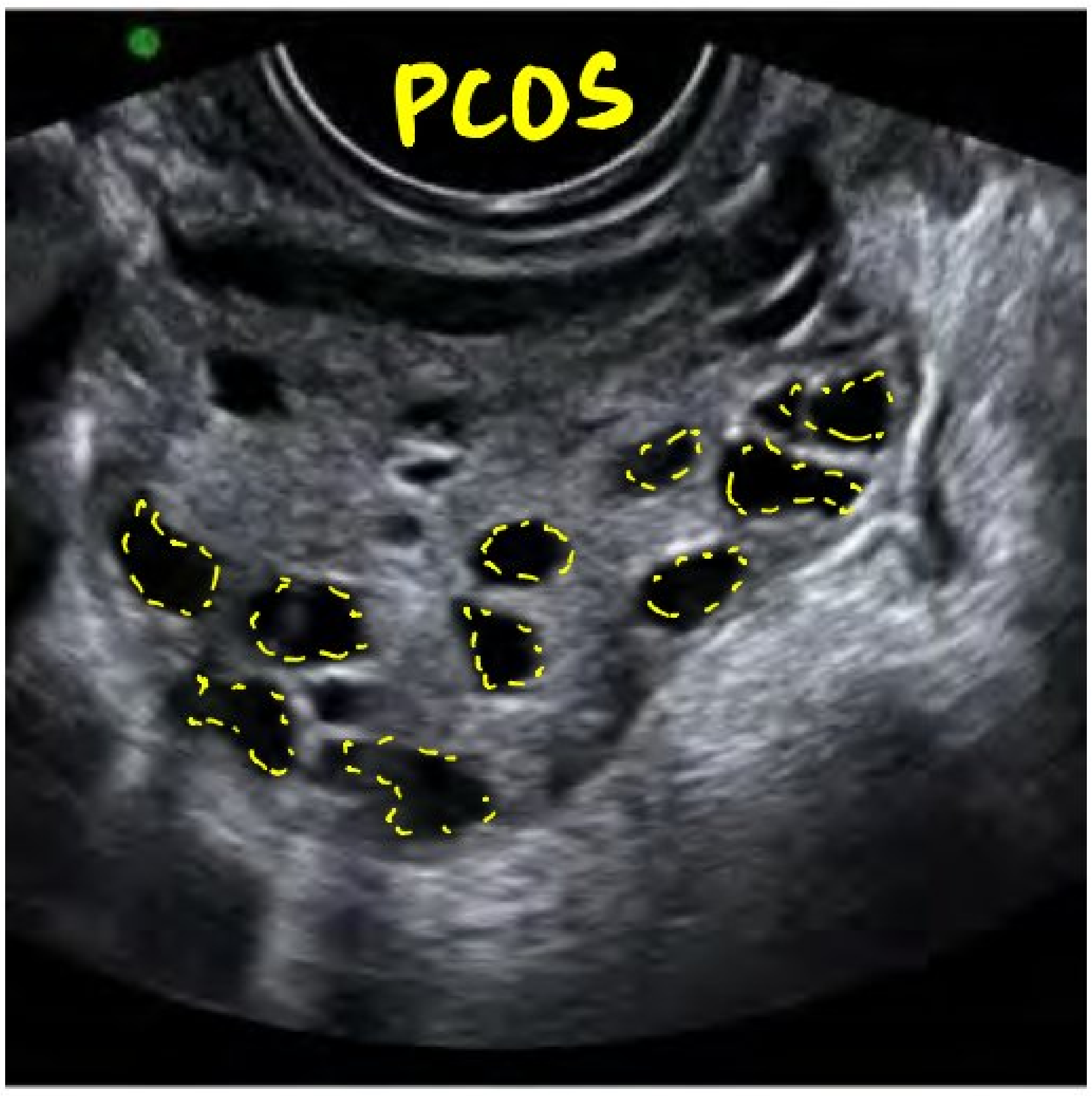
Peripheral arrangement – NECKLACE Pattern

↳ Characteristic of PCOS (not required for PCOS)

↑ Stroma (finding – Stromal Hyperplasia)

Thick capsule (Tunica)

Ovaries can be Ⓝ



D/D

Adult Onset CAH – milder form of CAH

↳ Closest D/D of PCOS. In every pt provisional diagnosis of PCOS – Rule out Adult Onset CAH.

LAB EVALUATION

1. S.Testosterone ↑ (< 200ng)
2. DHEAS (can be ↑)
3. USG Criteria
4. LH ↑, FSH – Ⓝ ; LH/FSH ↑ >2:1
5. PRL – Ⓝ
6. TSH – Ⓝ
7. IR – do OGTT
8. Lipid profile
9. Total Estrogen – ↑

   E_2 – Ⓝ
   E_1 – ↑
   E_2/E_1 Ratio is Reversed
   } S/i Obese PCOS

   Estrogen – Ⓝ in Thin PCOS
   Free E_2 – ↑

   HYPERINSULINEMIA

10. ↓ SHBG : Testis $\xrightarrow{\ominus}$ SHBG Synthesis ↗⊖
11. 17-OH Progesterone level – Ⓝ

Complications

LONG TERM

- Type-2 DM
- HTN
- CAD
- Endometrial/Ovarian Cancer
- Non-alcoholic Steato hepatitis
- Sleep Apnea Synd.

SHORT TERM

- Hirsuitism
- Infertility
- Irregular cycles

PSYCHIATRIC COMPLICATIONS

- Depression
- Anxiety

Pregnancy - High Risk of Abortion, GDM, P.E, Still birth, Pre-Term Labour.

Management

1. if Obese PCOS women → 1st advice → Wt loss (5-10%)

DOC - OCP - very low / low dose pills [4th Gen Progesterone used]
↳ Provide endometrial protection

2. If only HIRSUITISM - DOC → OCP
↳ 2nd line → SPIRONOLACTONE 3rd - CYPROTERONE Acetate

4th - Metformin - for Mild hirsuitism 5th - GnRH agonist - continuous

6th - Flutamide 7 - FINASTERIDE 8. KETOCONAZOLE

OCP - E+P → feedback inhibition of Ovary
↳ Given as long as possible

- DANAZOL - inhibits GnRH
↳ itself severe Androgenic S/E

- EFLORNITHINE - Topical Agent for Hirsuitism

Management of Infertility : ANOVULATION - 1st advice for obese → Weight loss

OVULATION INDUCTION

1st line - LETROZOLE >> CLOMIPHENE CITRATE

Aromatase Inhibitor

↑ FSH

5 days

[D5-9 or D2-6]

SERM

dose : Antagonistic action
↳ at which induction of ovulation

↑ FSH ; 5 days

Monofollicular development
No Risk of Twins

Live Birth Role is better c̄ LETROZOLE

- Start c̄ 2.5 mg ⟶ 7.5 mg
- 2nd Line : Inj. Gonadotropins (LH/FSH)

(initially made from urine of Post-menopausal women)

- Human Menopausal Gonadotropin (HMG) - 75 IU of FSH and LH

- because of presence of impurities less effective but COST EFFECTIVE
- Recombinant FSH → Effective, Expensive
- Risk of ① Multifetal Preg. - 30%
  (Higher Order Gestation)

② OHSS - 15%
↳ Highest c̄ Gonadotropins

IVF - (MC) used drug → Gonadotropins

Multifollicular development
↓
Multifetal pregnancy

(Twin Pregnancy @ maximum)

- NO RISK of Triplets/Quadriplets

Start c̄ 50mg $\xrightarrow{\uparrow}$ 150mg

(MC) S/E - Hot flashes

2nd (MC) S/E - Ovarian cyst

OHSS - Ovarian Hyperstimulation Syndrome } 1%

Clomiphene Citrate - 80% Ovulation but only 40% - Pregnancy
↓
makes
Endometrium - THIN

So, Antagonistic action of CC
↳ makes MUCUS - THICK

- Other drugs used for Ovulation Induction

3. GnRH Agonist - Pulsatile (Hypothalamic cause) - KALLMAN's Syndrome

4. In HYPERPROLACTINEMIA → cause Anovulation and Infertility

Bromocriptine > Cabergoline

INFERTILITY

Definition : Inability to conceive even after 12 months / 1yr of unprotected intercourse

aka SUBFERTILITY.

≥ 35yrs - beyond 6months - start evaluation

- Male factor - 40%
- Female factor - 40-45%
- Unexplained - 10%

- Female factor - (MC) Ovarian dysfunction → ANOVULATION
  ↳ OVARIAN FAILURE

TESTS OF OVULATION

1. Easiest Test - S. Progesterone level done on D_{21} of the cycle : if ≥ 3ng (Ovulatory)

2. Cervical mucus : E → P , Thick mucus Scanty

3. Vaginal cytology : P → INTERMEDIATE Cells
↳ (C) vaginal fornix - (Lateral wall)

4. Basal Body Temperature (BBT)
- ↑ 0.5°F
- BIPHASIC Chart

5. ENDOMETRIAL BIOPSY → Tissue Diagnosis
↳ Most INVASIVE
↓
Once at begining of evaluation for INFERTILITY - 2 Samples taken 1. HPE - Formalin 2. AFB - Saline

- done in Pre-Menstrual phase - 2-4 days before expected date of Menses D_{26} (24-26)

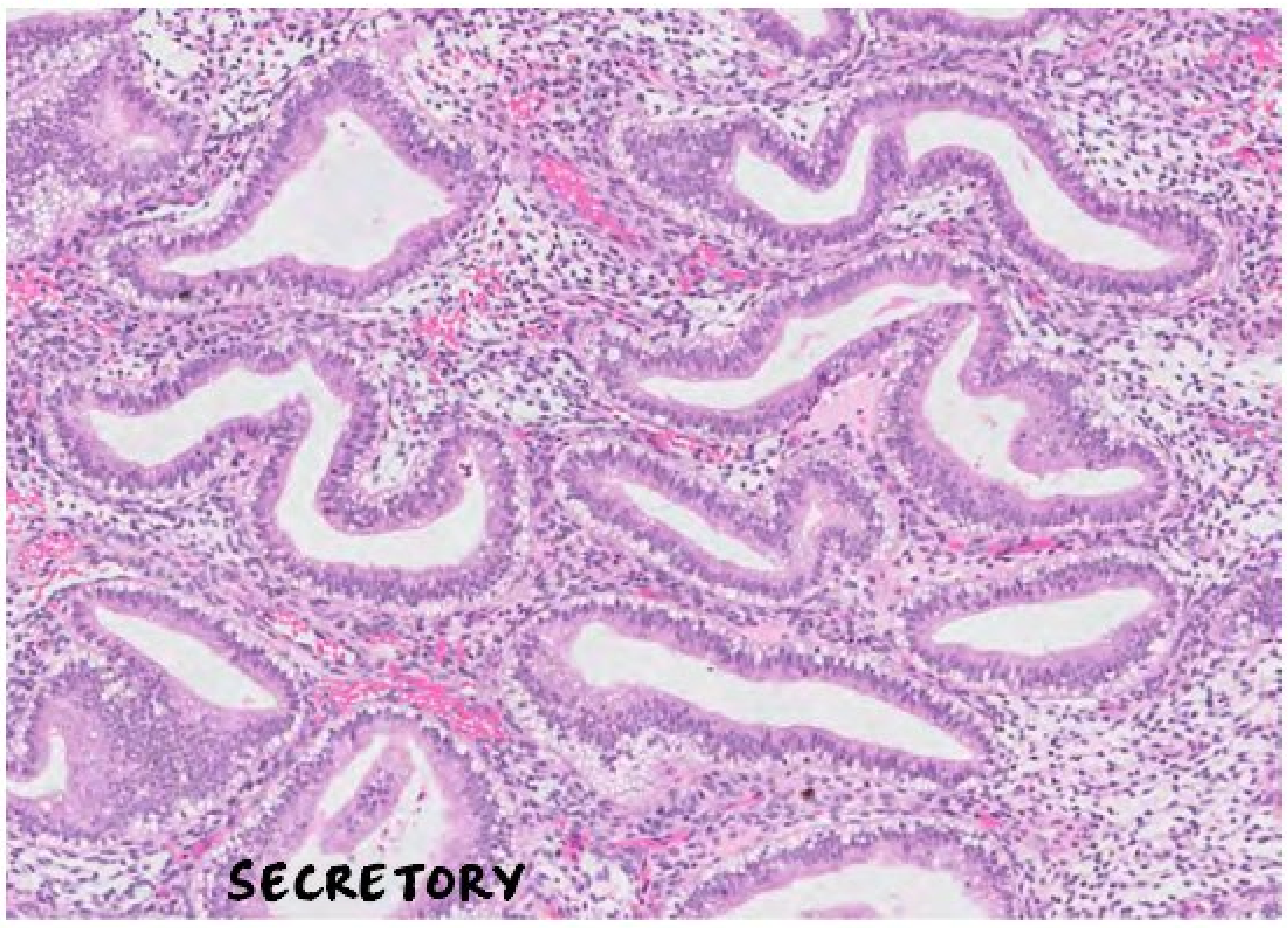
- HPE Report : Ovulatory - Secretory Endometrium

  Anovulatory - Proliferative

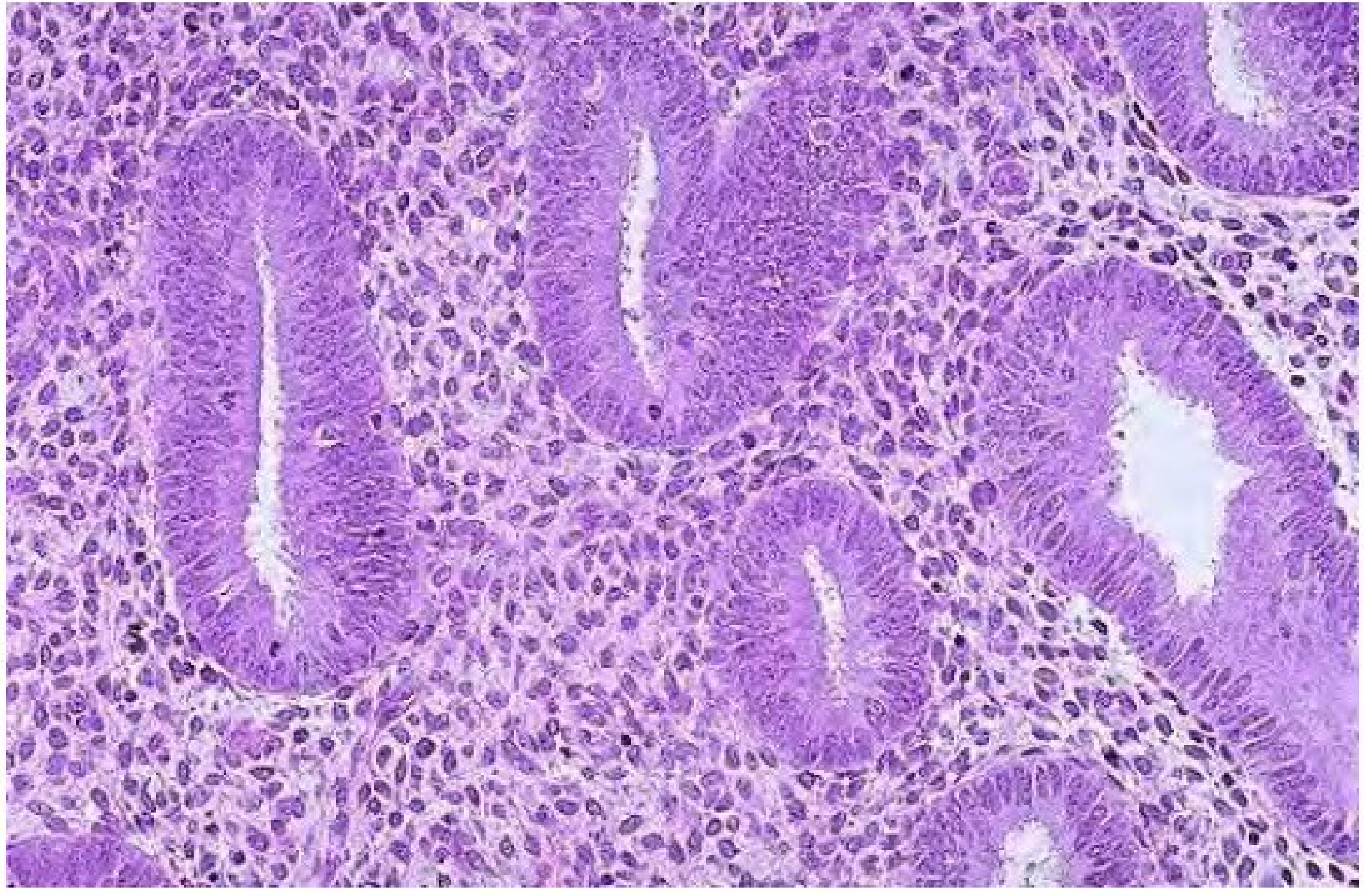
Glands - long; tubular → Telescoping of Endometrial glands
Nuclei - are at different levels - Pse dostratification
} Proliferative Endometrium
↓
↓ Effect of ESTROGEN

- SECRETORY PHASE : Cells have vacuoles → Subnuclear Vacuolation → Early Secretory Endometrium
  ↓
  1st HPE Evidence of Progesterone Secretion : D_{16}

- LATE SECRETORY PHASE : Coiled glands — CORKSCREW glands - Maximum Stromal Edema



Proliferative



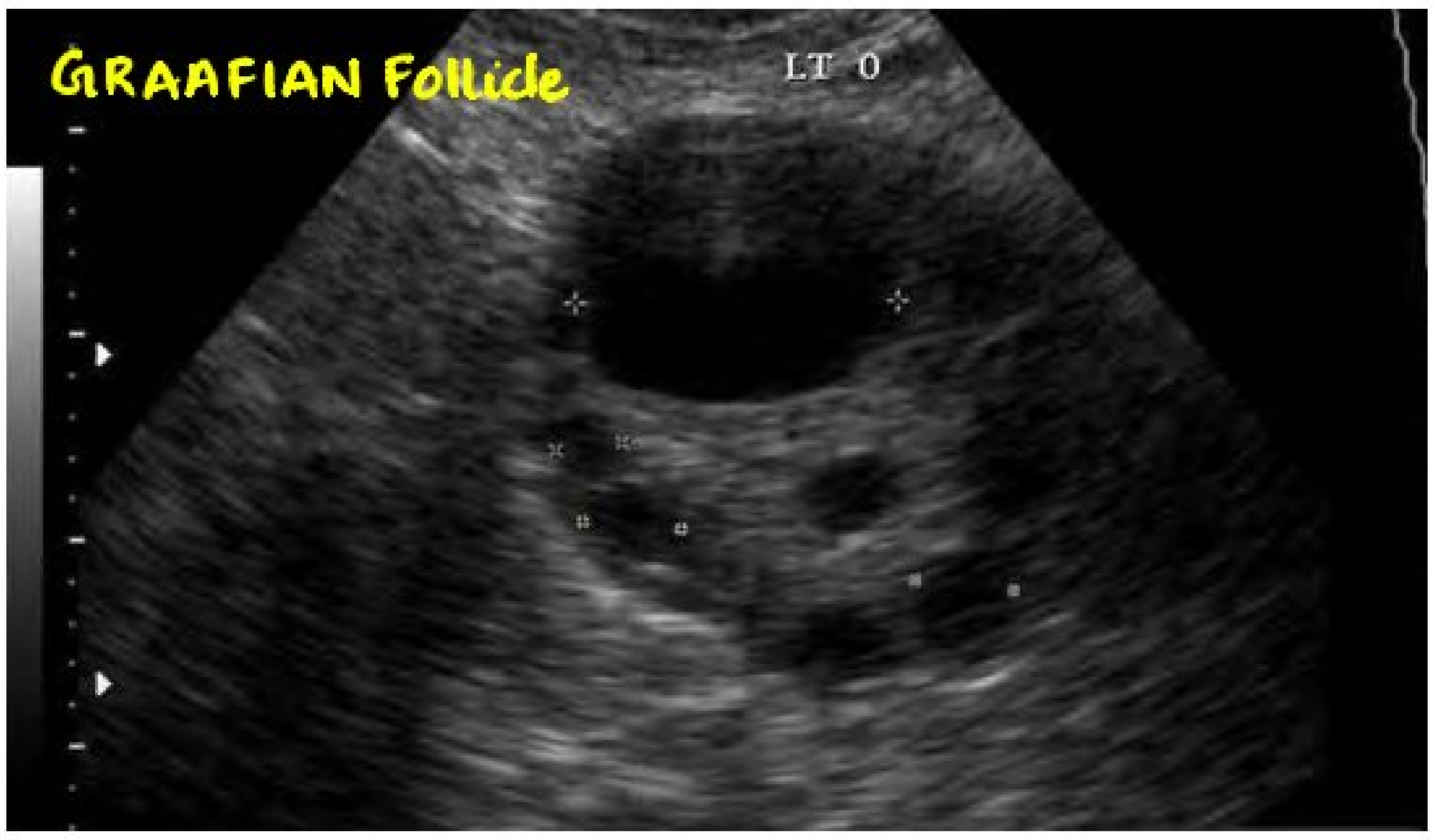
6. TVS for FOLLICULAR Monitoring (D_{10})

- Growth of Follicle → 2mm per day ;

18 - 20mm - GRAAFIAN Follicle / MATURE Follicle
- fluid filled cavity - ANTRUM

↓

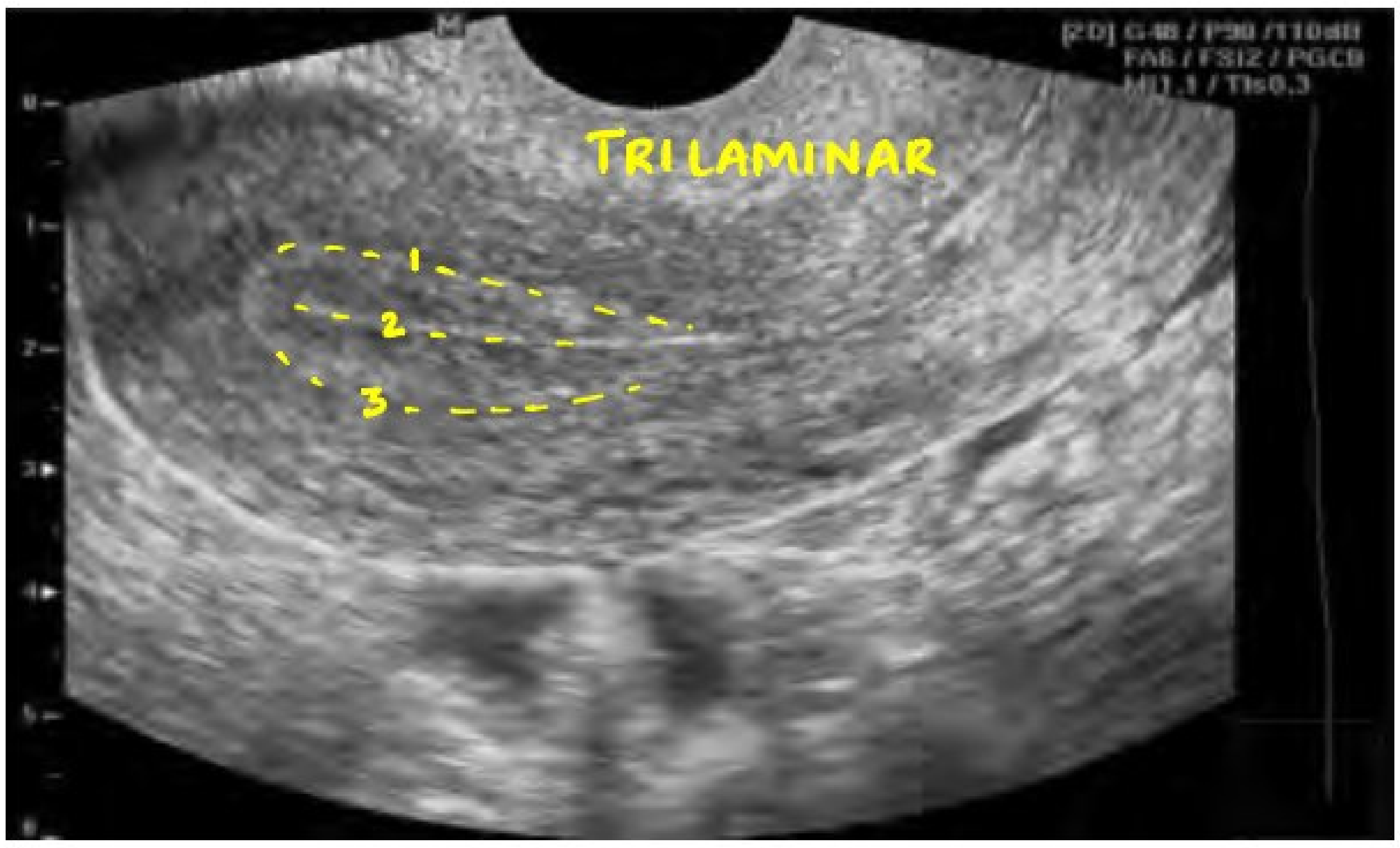
Rupture.
On USG - Sudden ↓ in Size
Fluid in the pouch of DOUGLAS



On USG : TRILAMINAR : Triple layer

Late Proliferative Phase
(Peri Ovulatory phase)

1. Anterior Endo-myometrium jn
2. Cavity
3. Posterior Endo-myometrium jn



On USG - Post. Enhancement - Secretory phase of uterus — Single Hyperechoic line c̄ posterior enhancement

7. URINARY LH Kits (LH Surge)

- urinary LH Surge $\xrightarrow{24\text{hrs}}$ Ovulation

TESTS OF OVARIAN RESERVE

1. (MC) used is S. FSH

Ⓝ < 10 I.U ,

if b/w 10-15 IU : BORDERLINE ≥15 IU : Poor Ovarian Reserve

≥ 20 IU : S/o Ovarian failure

done on Day 3 (D_2-D_4)

≥ 40 IU : Diagnostic of Ovarian failure

2. S. AMH (Anti Mullerian Hormone) - MIS

↳ Small Antral and Pre-antral follicles

Ⓝ = 1

if < 0.5 → poor ovarian Reserve

- if ≥ 3.3 - 3.5 → High Risk of OHSS

3. S. INHIBIN-B - directly comes from follicle (D_3). If < 45pg - Poor Ovarian Reserve

4. AFC (Antral Follicle Count) - TVS

- combined Score of Both ovary < 10 ; Poor Reserve

5. CCCT - Clomiphene Citrate Challenge Test : D_3 - FSH
D_{5-9} - 100mg CC
D_{10} - FSH

- High value on D_3 which rises further on D_{10} – Poor Ovarian Reserve

- Lap Ovarian drilling - destroy stroma (Burnt) ⟶ Ovary - Androgen production will ↓se

2nd Line Rx Gonadotropins >> Lap

Ovarian factor >> Tubal factor upto 35% (20-30)
↓
IOC : HSG - Screening test for Tubal patency

- done in Post-menstrual phase
- done b/w D_5 - D_{11}
- (MC) D_{10}

LEECH WILKINSON'S CANULA - inserted into cervix
↓
dye is inserted in the uterus
(water soluble Iodinated dye ~10ml)
↓
Serial X-Ray → PERITONEAL Spill

Contraindications

1. Pregnancy
2. K/c/o Gonadal TB
3. Discharge from Cervix and vagina
4. K/c/o Dye allergy

- Before doing HSG - UPT ⊖ ; Endo. Biopsy - AFB ⊖

- Abnormal Report : U/L Block or B/L Block B/L Hydrosalpinx produce B/L Distal Block

B/L Proximal Block : Better Prognosis ? Physiological - • pain (spasm)
• Mucus plug @ cornua
↓
Next do Diagnostic procedure
Diagnostic Laparoscopy ↓GA and Chemo pertubation
+
HYSTEROSCOPIC Canulation (Guidewire - plug gets destroyed)
↓
Therapeutic ⟶ Tubes are still BLOCKED : IVF >> Microscopic Surgery

B/L DISTAL BLOCK → Next step : Diagnostic lap + Chromopertubation → Mild
Severe

if severe ds → do IVF

- When there is Anatomical distortion - FROZEN PELVIS ⟶ S/i 1. TB genital
2. Endometriosis
3. Severe PID

eg: B/L Distal Block / B/L Hydrosalpinx Block → do IVF

- if Mild disease -
  1. do fimbrioplasty
  2. Adhesiolysis

- Fluid inside HYDROSALPINX is EMBRYOTOXIC
- Outcome of IVF in Hydrosalpinx can be improved by doing Salpingectomy before IVF

GENITAL TB

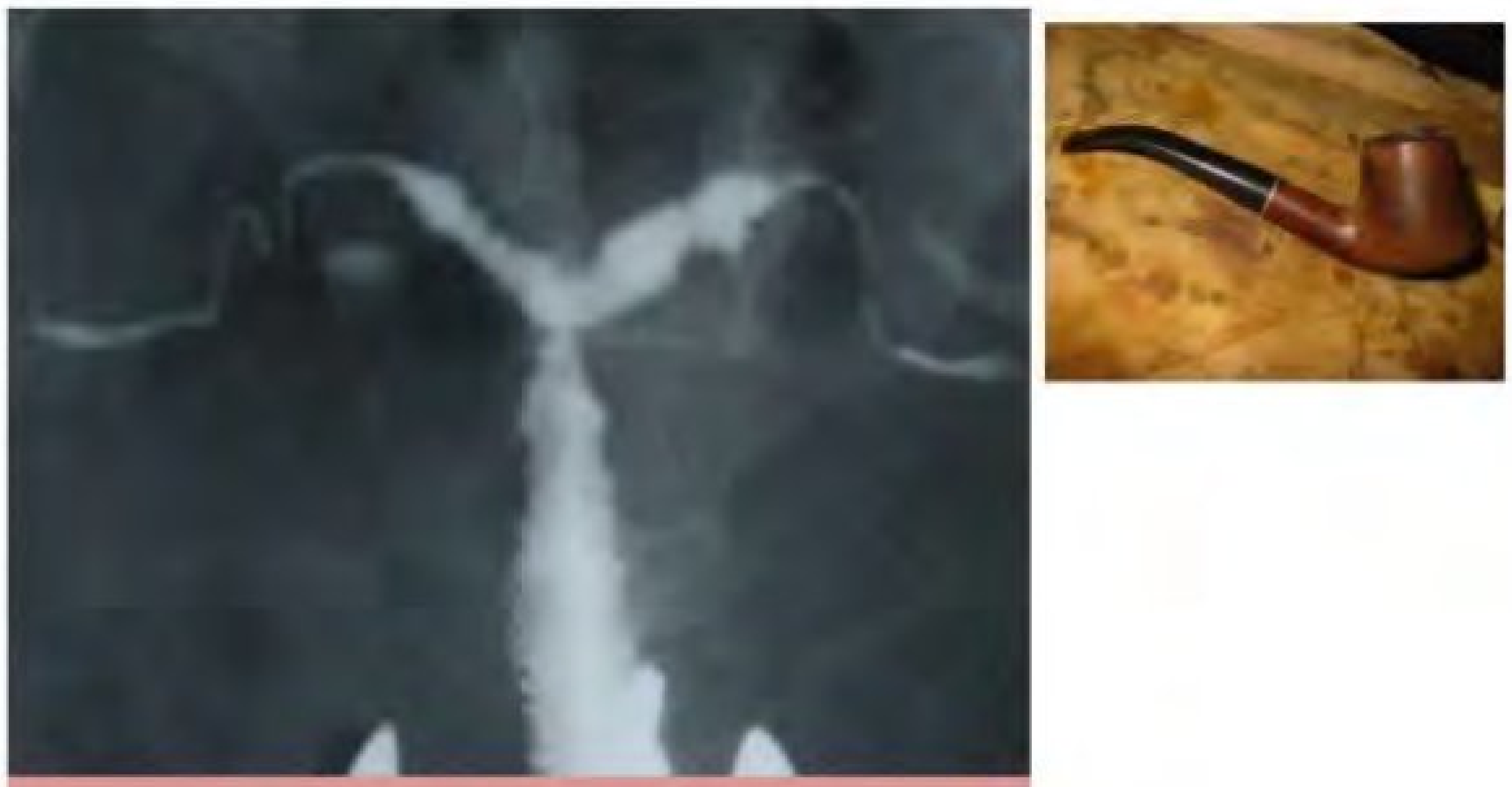
- 2° infection ((MC) 1° Lungs > Lymph nodes)
- (MC) mode of spread : Hematogenous ; others - Direct spread / Lymphatic / Ascending infections
- (MC) site : Fallopian tubes (>90%) > uterus (50-60%)
- (LC) site : Vagina and Vulva (<2%)

FALLOPIAN TUBE : (MC) Ampulla > Isthmus (spare medial part - INTRAMURAL)

- initially - Edematous inflammed
- Later - Fibrous (adhesions) / HSG

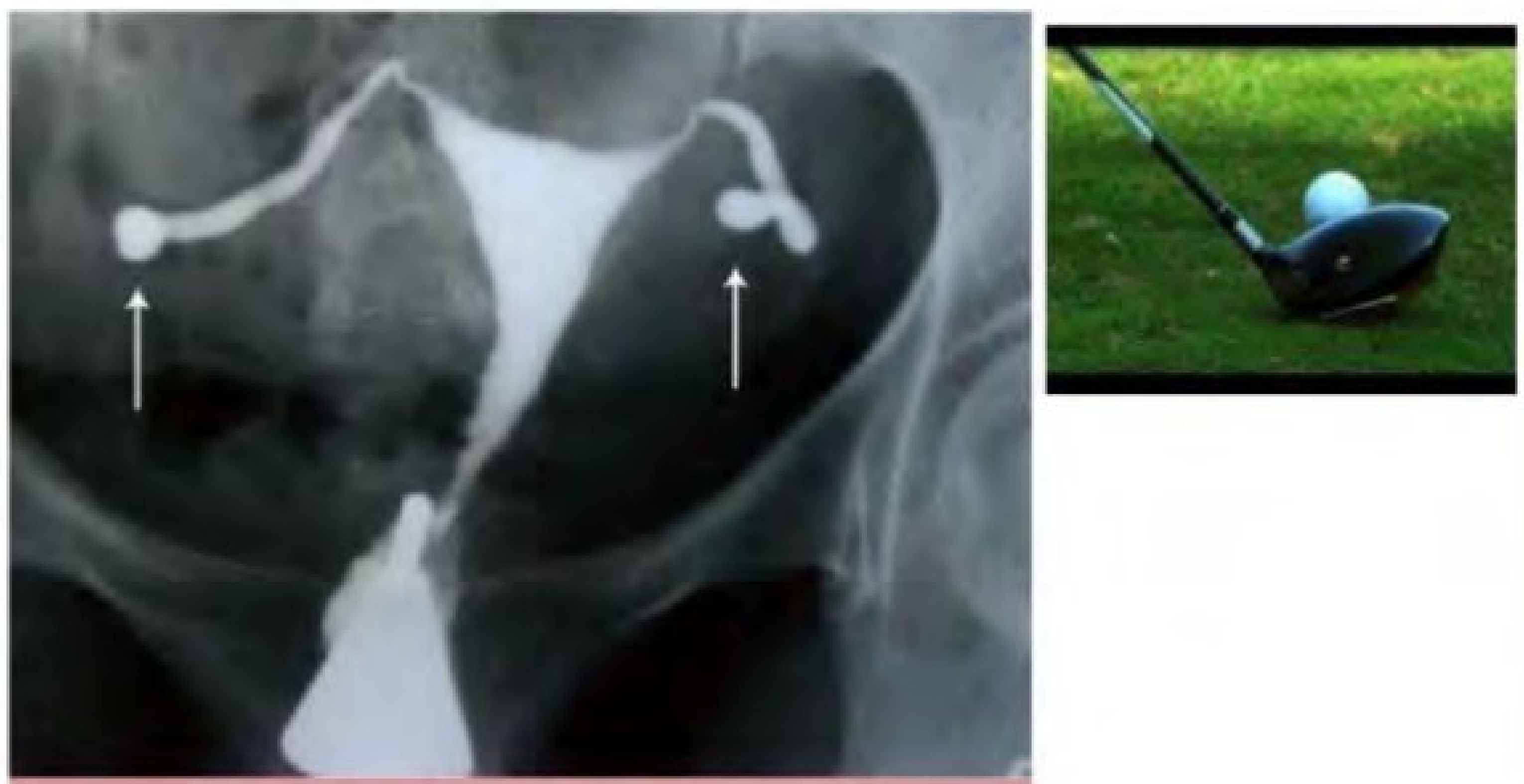
Named Signs s/o HSG

1. LEAD PIPE / PIPE STEM
2. BEADS ON STRING
3. COBBLE STONE App
4. GOLF CLUB App
5. TOBACCO POUCH



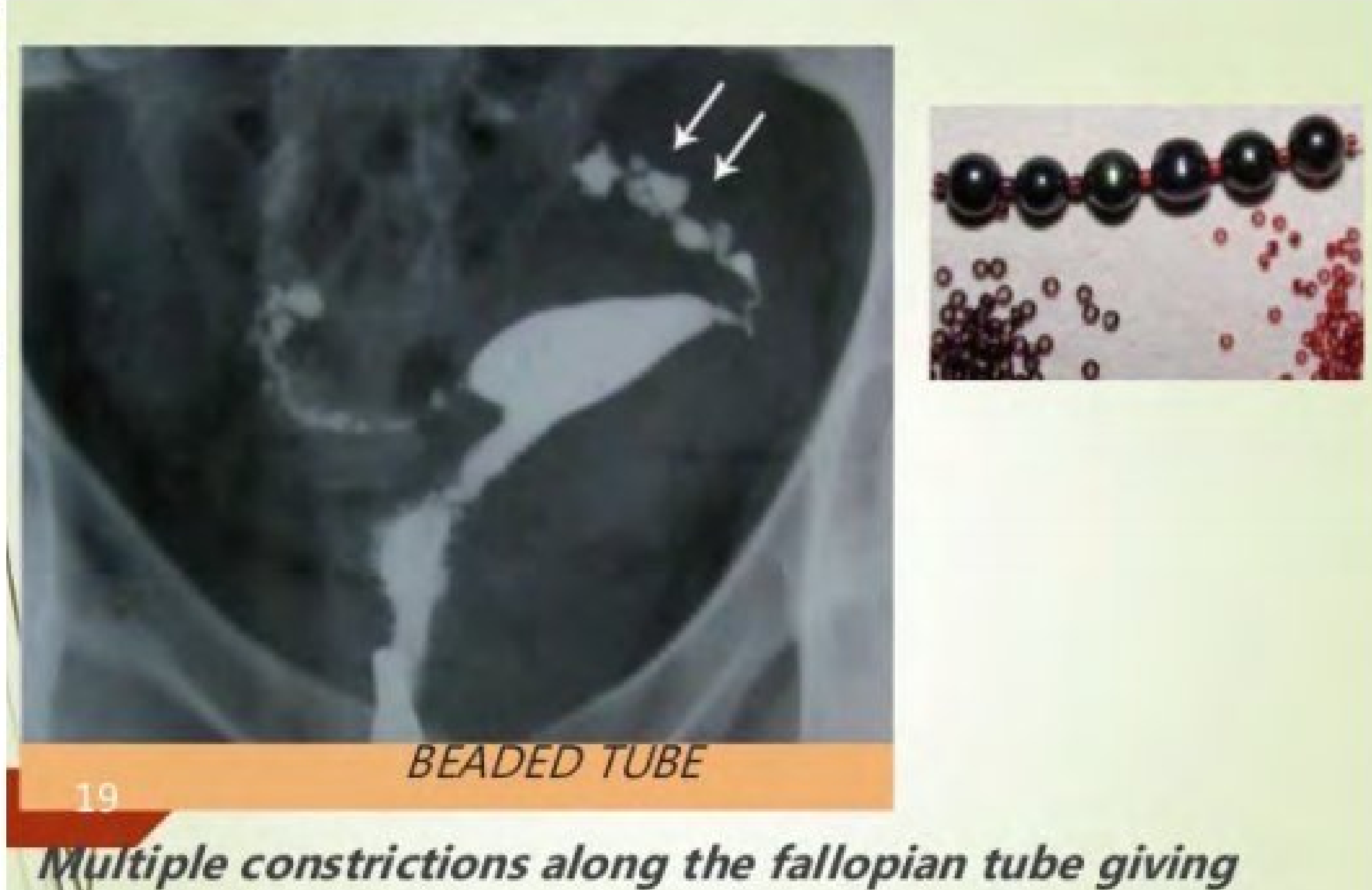
PIPE STEM APPEARANCE

Absence of normal tortuosity and a curved or straight pipe like appearance show fibrotic stage of tuberculous salpingitis. Irregular contour of the uterine cavity with diminished capacity in the fundual portion resembling a septate uterus.



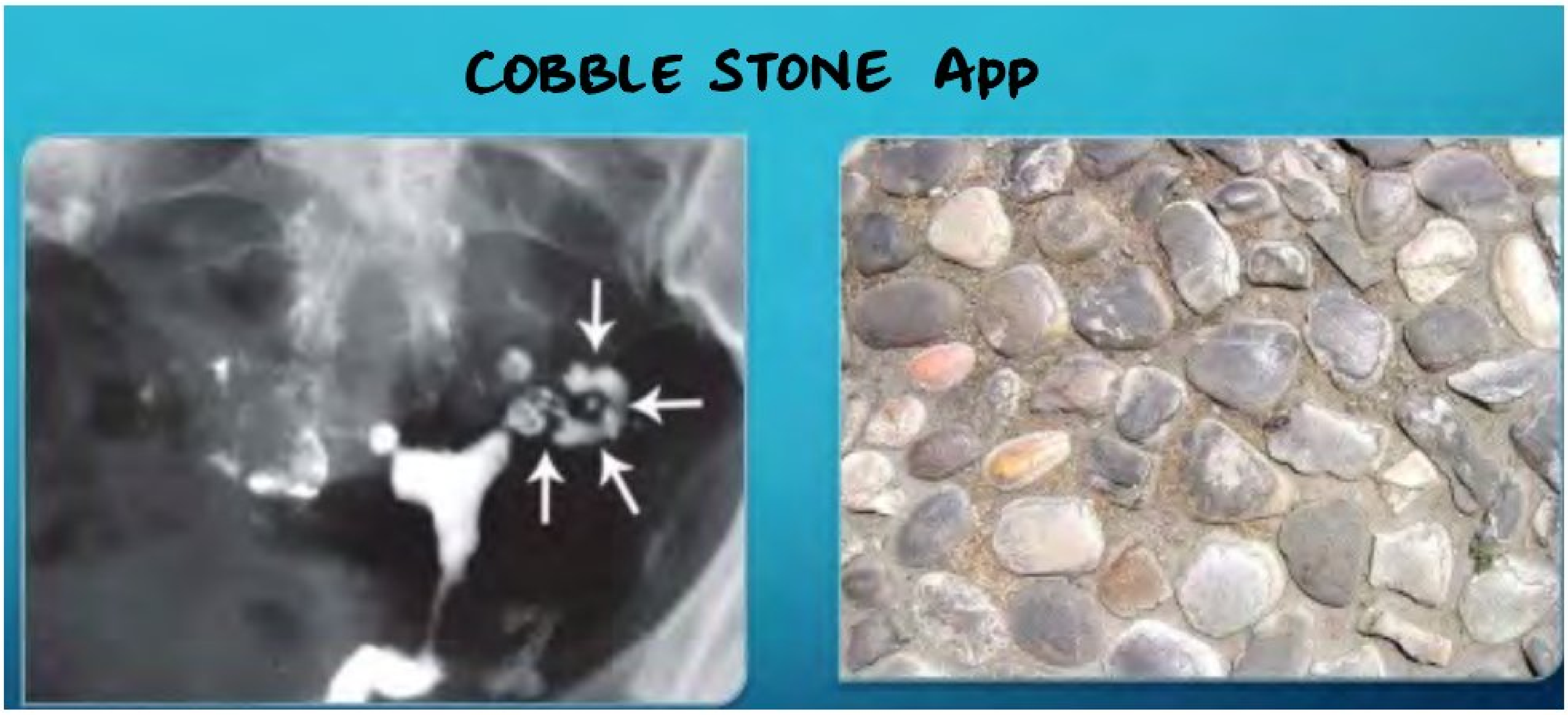
GOLF CLUB TUBE

Sacculation of both tubes in distal portion with an associated hydrosalpinx giving a Golf club-like appearance (arrows).



BEADED TUBE

Multiple constrictions along the fallopian tube giving rise to a " beaded" appearance [arrows]



- **TB uterus** - Only the endometrium (suffered)
  Myometrium - Spared
- Route of spread : DIRECT Spread
- (MC) presentation of Genital TB

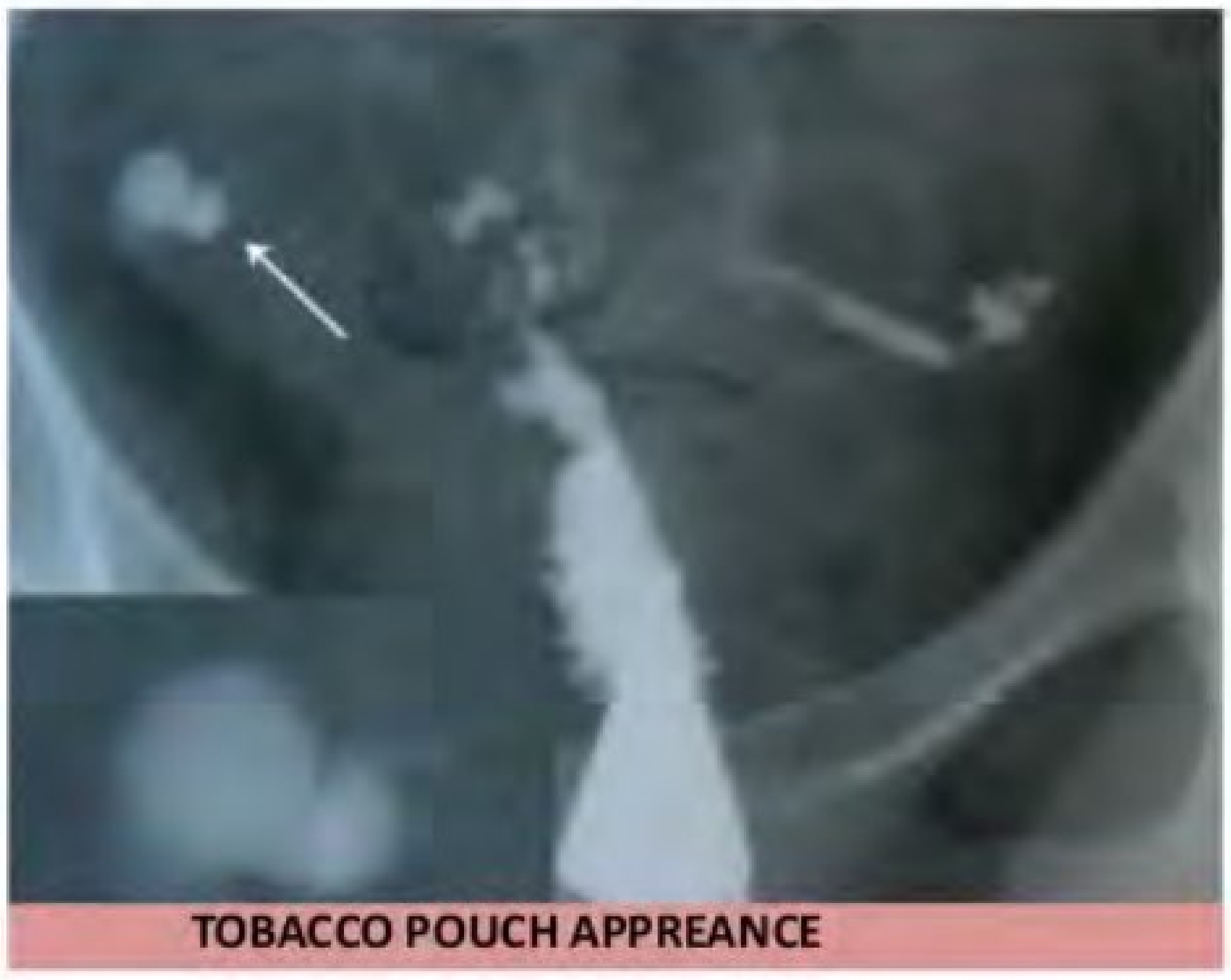
  Infertility f/b Pelvic pain f/b

  Menstrual irregularity

  ↓

  1st Menorrhagia (Endometritis)

(MC) Amenorrhoea > Oligomenorrhoea



Terminal hydrosalpinx with the conical narrowing is seen in the right tube (arrow). Eversion of the fimbria secondary to adhesions, with a patent orifice produces the tobacco pouch appearance in the left terminal.

(MC) Finding - (N) Pelvic examination → Tenderness in pubis

- in Reproductive age women : TB not present as B/L Adnexal masses
- Diagnosis : Best by Endometrial Biopsy - AFB ; culture

  Menstrual Blood for PCR Analysis for TB Analysis → 1st day sample

Rx: ATT × 6 months
↳ also improve fertility

In severe Genital TB (Anatomy is distorted) → Do IVF

UTERINE CAUSES OF INFERTILITY

- distortion of shape of cavity
- destroys the endometrium

1. Asherman's Syndrome

2. Endometrial Polyp

3. DiEthyl Stillbestrol ·
. pd 'T' shaped uterine cavity

also cause HYPOPLASTIC UTERUS

4. Acute Retroverted Uterus :

Malalignment b/w Uterus and Cervix

5. Submucosal fibroids

6. Endometritis

7. Mullerian anomalies - donot usually present as INFERTILITY
↳ usually presents as ABORTIONS

- INCIDENTICAL FINDINGS on HSG or Routine 2D USG → 1st Invg. done
- Both are Screening test
- IOC : 3D and 4D USG
- Gold Standard : MRI

- On HSG

1. ARCUATE uterus : Small dip at Centre . BEST Reproductive Outcome.

2. SEPTATE Uterus : ACUTE ANGLED, < 90° . WORST Reproductive Outcome.

3. BICORNUATE uterus : WIDE ANGLED

4. UTERINE DIDELPHYSIS : 2 uterus horns ± 2 vagina : a/w VAGINAL SEPTUM

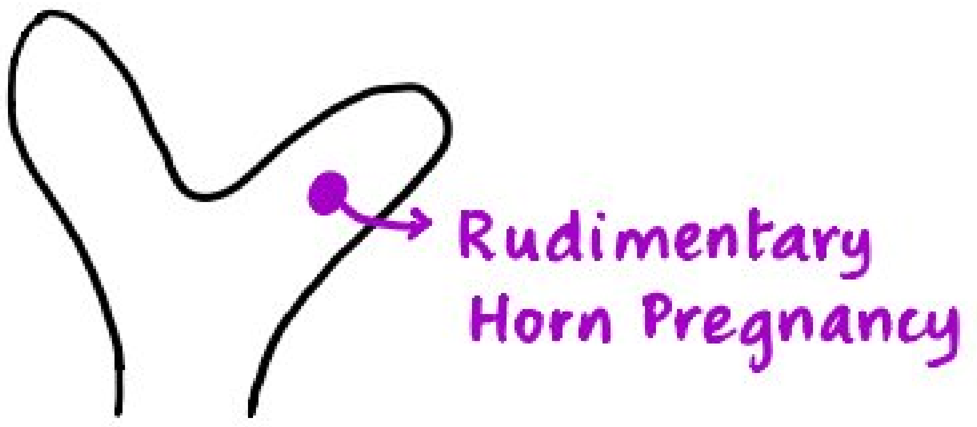
5. UNICORNUATE UTERUS

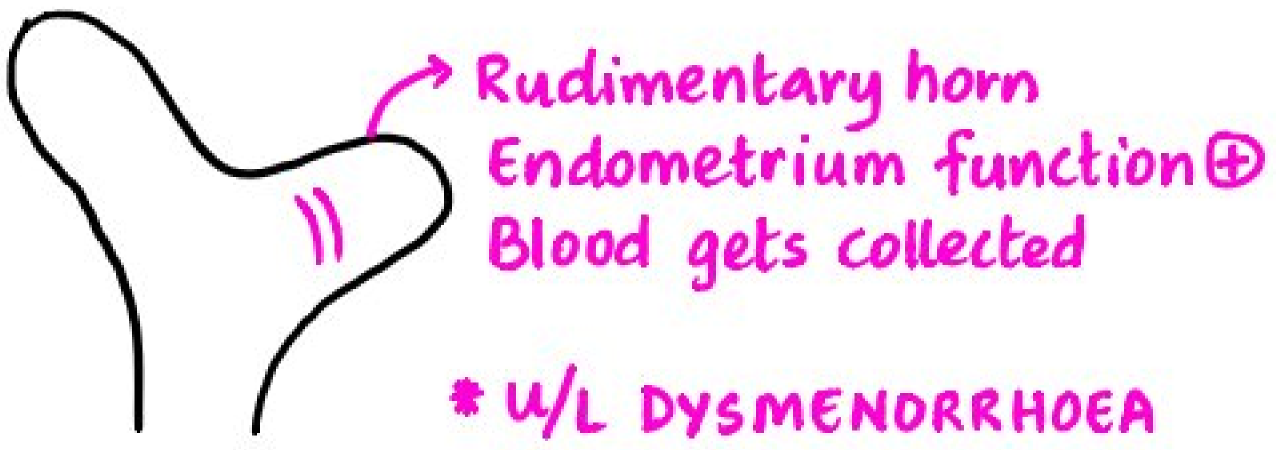
SEPTATE uterus : (MC) a/w ABORTIONS and INFERTILITY ; (MC) Mullerian Anomaly

BICORNUATE UNCOLLIS : 2nd (MC) Mullerian Anomaly

- BEST Reproductive Outcome : ARCUATE > UTERINE DIADELPHYSIS

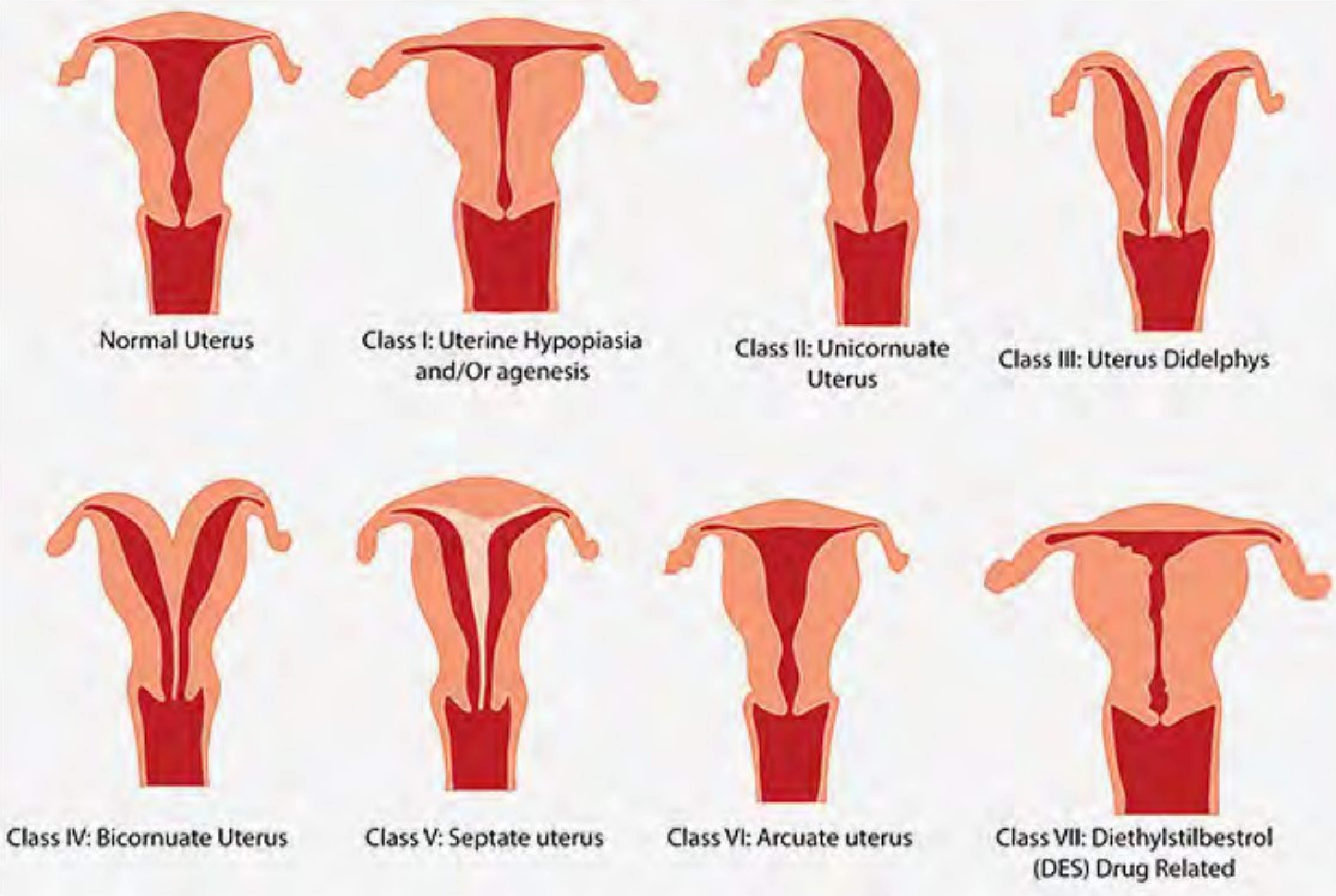
- High Risk for Urinary Tract anomalies : must undergo USG - Kidney and Bladder
  ↓
  Highest ≤ URICORNUATE UTERUS a/w ECTOPIC Pregnancy.





* U/L DYSMENORRHOEA

Endometrial polyps


Normal Uterus
Class I: Uterine Hypopiasia and/Or agenesis
Class II: Unicornuate Uterus
Class III: Uterus Didelphys
Class IV: Bicornuate Uterus
Class V: Septate uterus
Class VI: Arcuate uterus
Class VII: Diethylstilbestrol (DES) Drug Related

CERVICAL CAUSES OF INFERTILITY

: d/t MUCUS : can have ANTI-SPERM Ab (ASA) (IMMUNOLOGICAL INFERTILITY)

- Sperms ⟶ Cervical mucus ⟶ PROGRESSIVE Ⓝ = Forward
- Sperms ⟶ Cervical mucus c̄ ASA ⟶ ABNORMAL MOTILITY (circulatory / shaky motility)

POST COITAL TEST (PCT) - SIMS HUHNER Test

- Cervical mucus (Smear Post coitus) ⟶ Abnormal Motility
- usually done D_{12} - D_{14} - done for ASA

Sperm count ; sperm motility ; sperm morphology

New Test →
- IMMUNO-BASED Assay
- SPERM AGGLUTINATION Test
- SPERM MORPHOLOGY Test

} done for ASA

- NO Specific Rx for Antibodies. So, do IUI (Intra Uterine Insemination) → Bypass Cervix

Semen Sample → Processing ⟶ MEMBRANE FILTER can also be ⊕ in semen

More fertile semen ; ASA also ⊕ in the semen

INDICATIONS FOR IUI

IUI $\xrightarrow{OPD}$ Ⓧ APD (Assisted Reproductive Technology)

Maximum - 0.5 ml semen

1. Cervical Factor Infertility
2. Mild Male factor Infertility

- Sperms will reach fertilization site - Ⓝ Conception - 30mins

3. UNEXPLAINED Infertility : → Mx: CLOMIPHENE CITRATE + IUI (x 3cycles)

↑ Superovulation c̄

↓ if fails IVF

CC - 6months → 1st 3 months - Only C.C

→ Next 3 months - C.C + IUI

4. Minimal - Mild ENDOMETRIOSIS
5. SEVERE SEXUAL DYSFUNCTION → in Female - VAGINISMUS → Involuntary contraction of Vaginal muscles → PAIN

→ in Male - failure of ERECTION

MALE FACTOR INFERTILITY

Husband Semen Analysis

- Best way to collect sample - MASTURBATION, reach the lab c̄ in 60 mins
- Period of Abstinence - 2days - 7days
- Analysis done after LIQUEFACTION
- If Abnormal Report → Repeat Analysis (minimum gap b/w 2 reports 1wk)

WHO PARAMETERS (Minimum - Required for Ⓝ Conception)

1. Volume - ≥ 1.5 ml
2. pH - ≥ 7.2
3. Total Sperm Count - ≥ 39 million / Ejaculate
4. Sperm concentration - ≥ 15 million / ml
5. Total motility - > 40%
6. Progressive Motility - > 32%
7. Morphology - 4% [Strict TYGERBERG Criteria]
8. Vitality - 58%
9. WBC Count - < 1 million / ml

Most Important : MOTILITY > Sperm Concentration > Morphology

AZOOSPERMIA : Absence of Sperms

ASPERMIA : Absence of Ejaculate

ASTHENOSPERMIA : ↓ MOTILITY

TERATOSPERMIA : ABNORMAL Morphology

NECROSPERMIA : Dead sperms

GLOBOSPERMIA : Sperms c̄ ROUND Head (lack ACROSOMAL CAP)

In KLINEFELTER'S SYNDROME - Testes small and HYPOPLASTIC - Azoospermia and Infertility

KARTAGENER Syndrome - ASTHENOSPERMIA

CYSTIC FIBROSIS - AZOOSPERMIA (obstruction to flow of Sperm)

If AZOOSPERMIA → Repeat Analysis : CONFIRM AZOOSPERMIA

↓ Ⓜ︎Ⓒ cause of Male Infertility : defective Spermatogenesis at Testis (1° HYPOGONADISM)

do HORMONAL ASSESSMENT

LH / FSH / Testosterone

↓ if Ⓝ, then obstruction to flow of sperms - OBSTRUCTIVE AZOOSPERMIA

↓

USG < SCROTAL / TRUS

SEMINAL VESICLE : main contribution VOLUME

↓

Dilated Seminal vesicles ; Ejaculatory duct obstruction

Absent Seminal vesicles ; CYSTIC FIBROSIS ; Absence of Vas deferans

RETROGRADE Ejaculation (↓ volume of Semen) ⟶ Assessed by POST-EJACULATORY URINARY ANALYSIS

If HORMONES - AbⓃ

↓ 1° Hypogonadism → LH/FSH - High / TESTOSTERONE - Low ⟶ NON-OBSTRUCTIVE Azoospermia

↓

do TESE [Testicular Sperm Extraction]

↓

TESE + ICSI ⟵ Non-motile

Intra Cytoplasmic Sperm Infection

if Obstructive Azoospermia : Rx - RESECTION and ANASTOMOSIS

IVF : Indications

1. Tubal factor
2. Male factor
3. Unexplained Infertility
4. Moderate - Severe Endometriosis
5. Ovarian failure / Gonadal Dysgenesis
6. Asherman's Syndrome
7. Mulerian Agenesis

MINIMUM SPERM COUNT Required.

- If 10 - 15 Million / ml - do IUI
- If 5 - 10 million / ml - do IVF
- If < 5 million / ml - do ICSI

8. PIGD - Pre-Implantation Genetic Diagnosis

- it is done when either Male/ Female Partner is K/c/o Genetic ds
- we take the embryo and test it for disease
- Genetic Material Used : 1. BLASTOMERES
  - Biopsy done on D_3 (8 cells)
  - usually we take 1 cell (maximum 2 cell)

2. BLASTOCYST TROPHOECTODERM
   - Better
   - done on D_5

3. POLAR BODIES DIAGNOSIS

IVF STEPS : COH (Controlled Ovarian Hyperstimulation Syndrome) - Ovulation Induction

- Gonadotrophins - Monitor : 1. Follicle count 2. $S.E_2$ levels

(Ⓝ Response : 1 follicle = 200pg of $S.E_2$)

Monitor ⟶ 4 follicles ; 18 mm ; $S.E_2$ = 800pg

- to Rupture → Inj. HCG - Ovulation trigger in IVF Cycle

↓ 3hrs After Injection

Ovum picked up : USG Guided.

↓

Embryo Transfer : (MC) done on D_3 - 8 cell stage
2cm below the fundus

Very High levels - High Risk:

1. ≥ 20 follicles ; > 10 mm
2. $S.E_2$ - > 3500pg
3. AMH - ≥ 3.3 - 3.5
4. PCOS
5. Pregnancy

OHSS - cause

↓

Inj. HCG → Inflammatory fluid : Mediators of OHSS - VEGF → Endothial injury

↓

Hemoconcentration ← Capillary leak
END ORGAN damage

No Specific Rx ⟶ Symptomatic Rx Only

Prevention :
1. Low dose gonadotrophins
2. Intensive monitoring → follicle, $S.E_2$

COASTING
3. Stop further gonadotrophins
4. Delay Inj. HCG
5. GnRH Antagonist

6. CRYOPRESERVATION
7. BROMOCRIPTINE (reducing severity of OHSS)
8. i.v Albumin (volume expander)

- Early Onset OHSS - ≤ in 9 days of Inj. HCG
- Late Onset OHSS - > 9 days of Inj. HCG

ENDOMETRIOSIS

- Endometrial glands and Stroma outside the uterus
- (MC) site : Ovary > Pouch of Douglas > Post. leaf of Broad ligament > Uterosacral ligament > Fallopian Tube

THEORIES

1. SAMPSON's Theory : Retrograde menstruation (most accepted theory)

2. MULLERIAN METAPLASIA Theory : by MEYER ; Theory of Coelomic metaplasia

3. GENETIC Theory - KRAS gene, GALT gene. 1st degree has Endometriosis - 7 times higher

4. IMMUNE Theory - have deficient cell mediated and Humoral immunity

5. HALBAN's Theory - Lymphatic spread ; Umbilical site Endometriosis

- Endometrial tissue - ESTROGEN - Reproductive Age Woman : 25-35yrs

Rare : Pre pubertal (Adolescent)
Post menopausal

In Pregnancy - PROGESTERONE ↑ ⇝ Symptoms will Regress

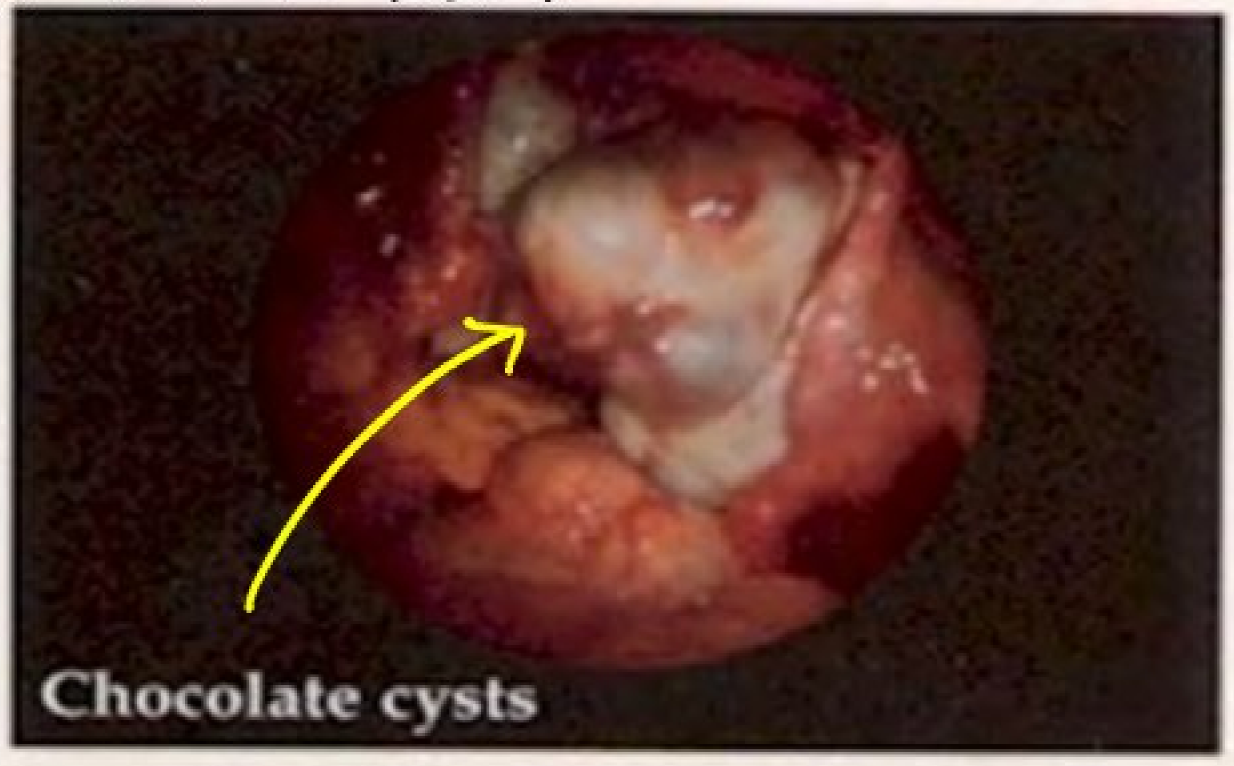
IOC : LAPAROSCOPY → 1. Diagnostic
↙
HPE (Biopsy)

2. Stage → Based on ASRM →
1. Minimal
2. Mild
3. Moderate
4. Severe

3. Surgical Mx of Pain

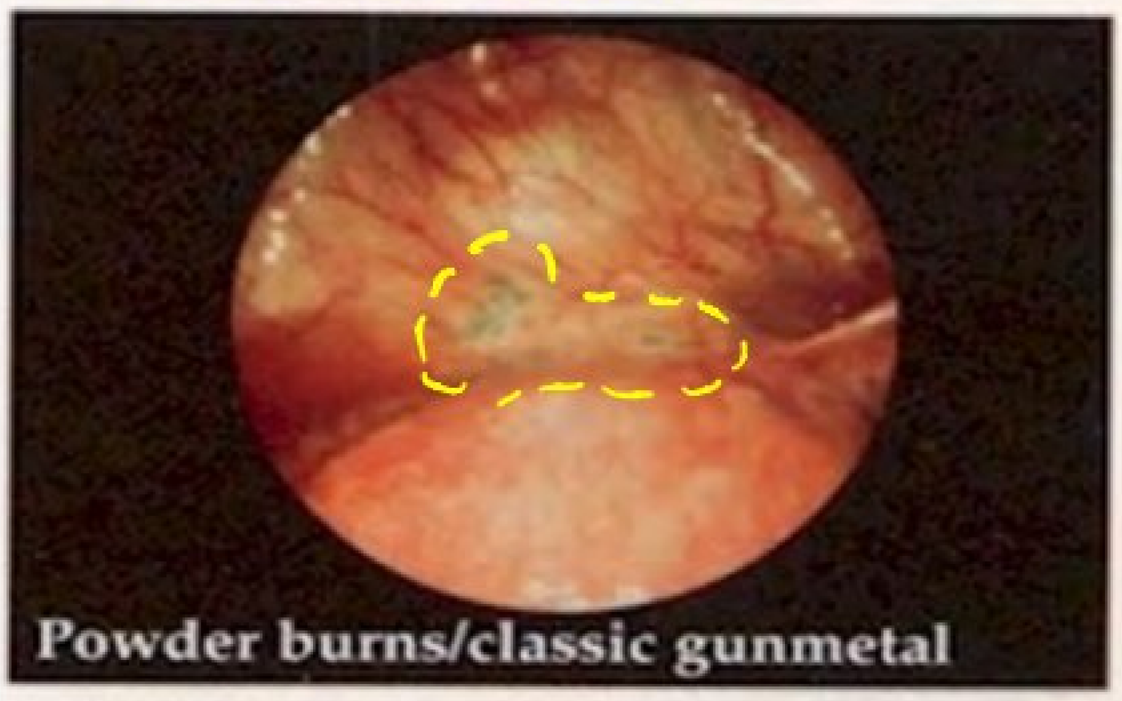
- RED FLAME lesion - S/o Fresh bleed (RASPBERRY or BLUESPOTS)

- POWDERBURNT lesion - S/o Chronic lesion
- Chocolate cyst c̄ Adhesions
- Peritoneal defects



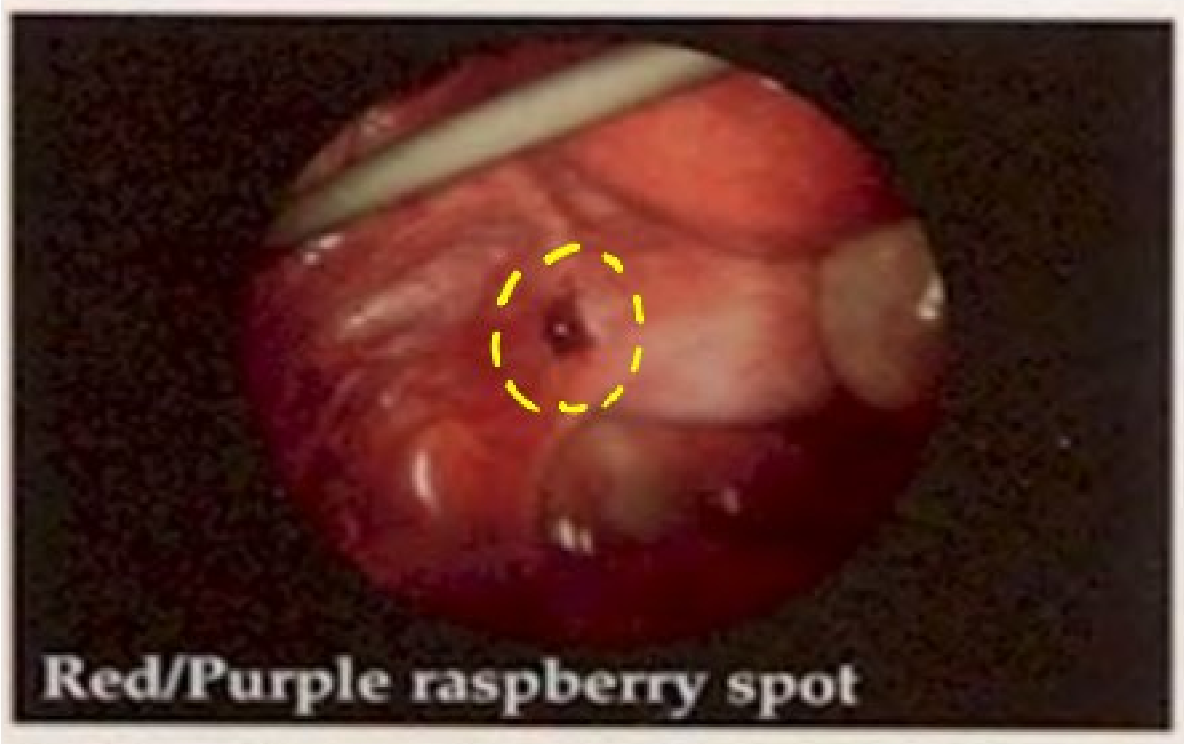
Chocolate cysts

Two chocolate cysts on the right ovary.



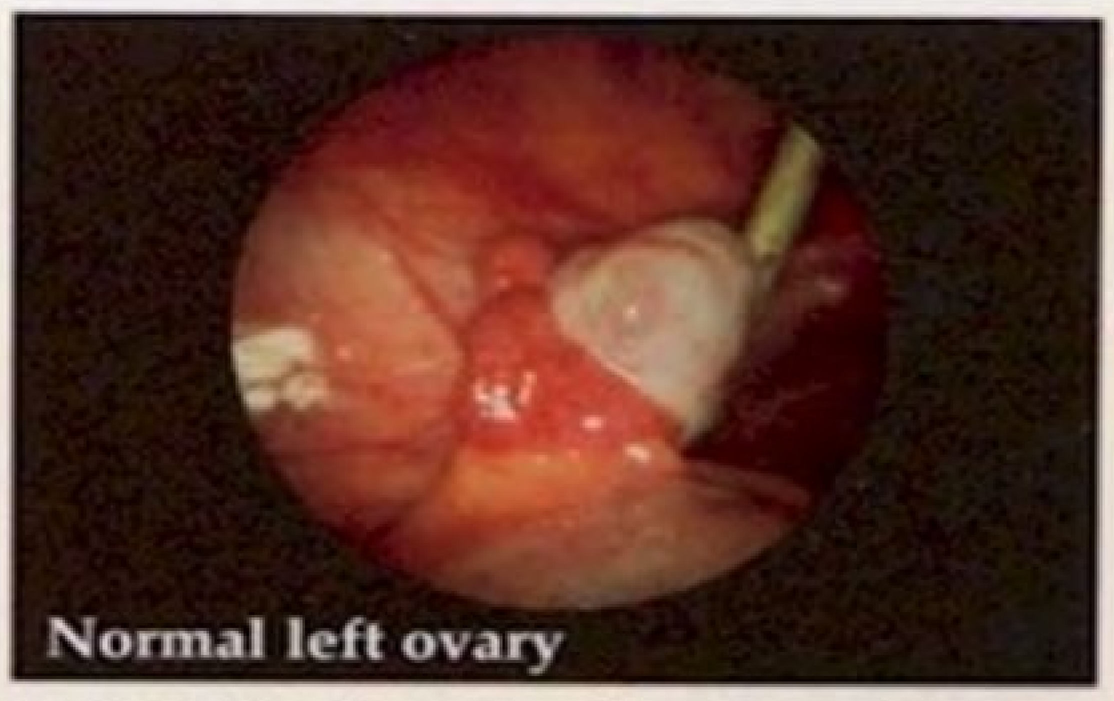
Powder burns/classic gunmetal

Dark lesions of endometriosis. These have been called blue-black lesions, gunpowder burns, and gunmetal lesions.



Red/Purple raspberry spot

Red and purple raspberry spot on the left uterosacral ligament.



Normal left ovary

Soft fluffy fimbria of a normal left fallopian tube.

Based on ASRM :

1. **MINIMAL** - Isolated lesions - **SUPERFICIAL**
2. **MILD** - Multiple lesions superficial aggregate - dia < 5cm
3. **MODERATE** - deep lesions (> 5mm deep in the peritoneum)
4. **SEVERE** - Anatomical distortion - • plenty of adhesions ⊕ • **CHOCOLATE CYST**

On USG :

CHOCOLATE CYST (ENDOMETRIOMA)

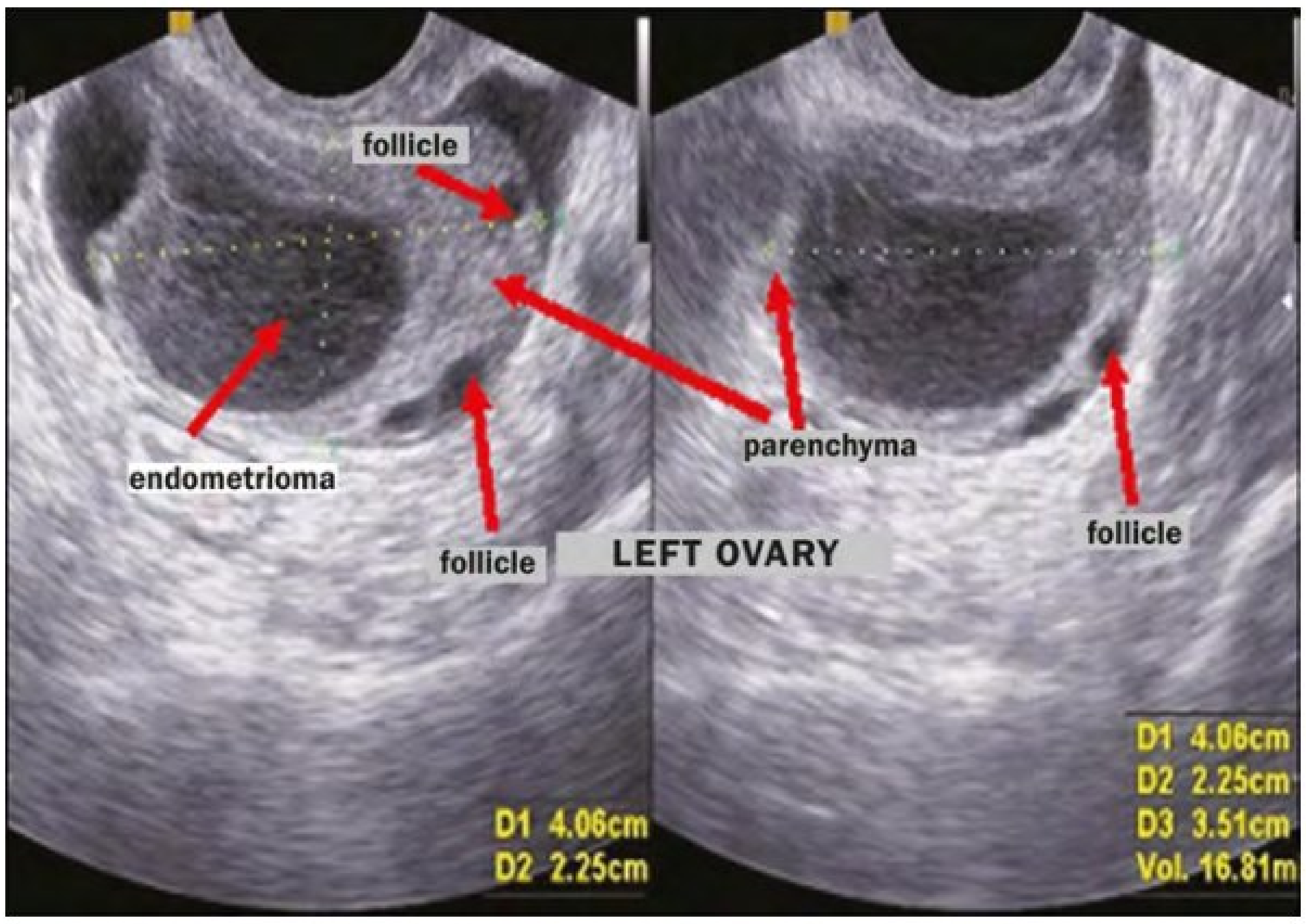
- cyst ≤ homogenous Internal Echoes
- Ground Glass App
- nodules on Uterosacral ligaments

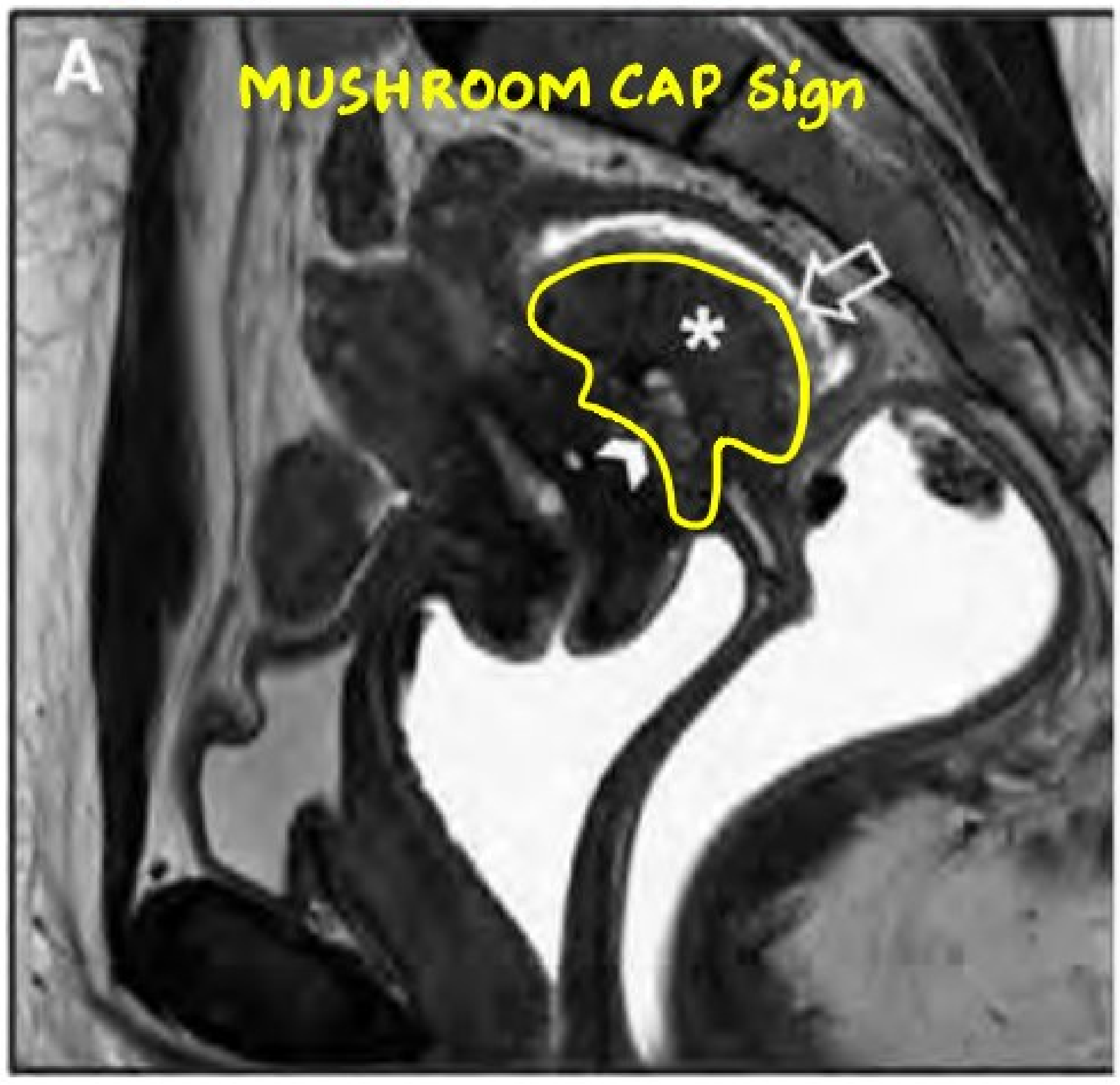
On MRI : Only under Special Required

↓ such as eg: MRI - Rectosigmoid - **MUSHROOM CAP Sign**

also done in ① **Deep Endometriosis**

② **Mullerian Anomalies**





CA-125 - ↑ upto 100 (Ⓝ < 35) → Non-specific marker in Reproductive Age Group

(MC) Presentation → **DYSMENORRHOEA** f/b **C/c Pelvic PAIN** f/b **DYSPAREUNIA**

Other presentation :
1. Adnexal mass
2. Infertility
3. Menstrual Problems (Menorrhagia)
4. **CATAMENIAL PNEUMOTHORAX**
5. **CYCLICAL HEMATURIA** (Bladder Endometriosis)

O/E :
1. Tenderness in Pouch of Douglas
2. Tender nodules on Uterosacral Ligaments → **BANJO STRINGS**
3. **FIXED** Retroverted Uterus

DYSMENORRHOEA

PRIMARY

- Physiological
- Spasmodic
- Pain in Suprapubic Region
- Pain begins on the 1st day of menses
- Improves c̄ menstrual Blood flow
- Disappear by 3rd day
- very Responsive to NSAID's

SECONDARY

- Underlying ds (MC) Endometriosis f/b PID
- Constant pain (Congestive Dysmenorrhoea)
- doesnot improve c̄ Menstrual Blood flow
- Persist - post menstrually
- less responsive to NSAID's

PAIN

① c/c Inflammatory Mediators: Endometrial deposits are made up of Endometrial tissue They will undergo Cyclical change and will bleed, there is release of Inflammatory mediators like PG's.

② More pain sensitive NERVE endings - aka NEUROMUTILATION (done by Estrogen)

- severity of pain depends on depth of implant

2° DYSMENORRHOE - Management of PAIN

Minimal - Moderate Pain - NSAID's + OCP's → given continuously 3-6 months

feedback inhibition → Suppress Ovarian Steroid production

↓ if no Response

DINOGEST (Progresterone) - continuous : oral ; im ; Mirena
- Thinning of Endometrium
- Implants - decidualization

↓

SEVERE PAIN : put her on GnRH agonist - given continuously (>5 pulses/hr, Ⓝ 1 pulse/hr)

↓ if No Response

Do LAPAROSCOPY : Surgical Management of Pain

1. Adhesiolysis
2. Fulgration of Implants
3. LUNA (Lap. Uterosacral Nr. Ablation)
4. OVARIAN CYST Rx.
5. HYSTERECTOMY - Last Resort Rx if family complete

POST SURGICAL : Suppression given in the form of OCP [AROMATASE INHIBITORS] DANAZOL - poorly tolerated]

CHOCOLATE CYST : doesnot respond to Medical management

↳ Rx.OC : CYSTECTOMY   ASPIRATION - High Recurrence Risk

Size: < 5cm - wait/watch

> 5cm - Symptomatic → CYSTECTOMY

INFERTILITY :

Minimal - Mild : CC + IUI × 3 cycles $\xrightarrow{\text{if fails}}$ IVF   Moderate - Severe : IVF

↳ Ovarian Dysfunction (Folliculogenesis Defective)

(MC) site : Ovary → c/c Inflammation

CERVICAL CANCER

- (MC) cancer Among Indian Women : CA BREAST
- 2nd (MC) - CERVICAL CANCER

Infection [HPV] ⟶ persist ≤ ↑ Age ⟶ Pre-Invasive lesions

- Regress spontaneously as well

↓

Infection → Time : 10 - 30yrs → Long h/o → INVASIVE

UNIVERSAL SCREENING : FIGO / ASA

- Start screen at 21yrs
- 21 - 29yrs - Only PAP smears → done 3 yearly
- > 30yrs - $\underbrace{\text{PAP + HPV}}_{\text{CO-TEST}}$ → done 5yearly
- 65yrs - STOP Screening (provided PAP smear in Last decade are NORMAL)

↓ if not Ⓝ - continuous screening

upto 75yrs

HPV - Higher sensitivity
PAP - Higher specificity

WHO SCREENING : starts at ≥ 30yrs ; CONCEP : Screen and Treat

- HPV > VIA > PAPs ↳ Ideal : 1st confirm and then Rx

HPV ↓ 5 yearly

VIA ↓ Visual Inspection c̄ Acetic Acid

VIA and PAP } 3-5 yearly

HPV + VIA >> HPV ↓ 3rd and 4th decade women

- HPV Test ⊕ve → Infected c̄ High Risk HPV but doesnot tell about Strain

  ↓ Hybrid Capture - COBAS → tells about strain

- HIGH RISK → Type 16, 18 → 70% of all Cervical cancer 50% ↶ 16 > 18 ↷ 20%

  Other strains (15) - High Risk viruses

- (MC) Strain TYPE 16 - cause SQUAMOUS CELL CA

- Most Specific TYPE 18 - cause ADENOCARCINOMA CERVIX

- HPV Virus - Viral proteins that cause CANCER : E_2, E_6, E_7

- HPV E_2 Ⓝ $\xrightarrow{\ominus}$ E_6, E_7 ; Viral genome INTEGRATE c̄ human genome. E_2 altered upregulate E_6, E_7 ⊗ → RB gene

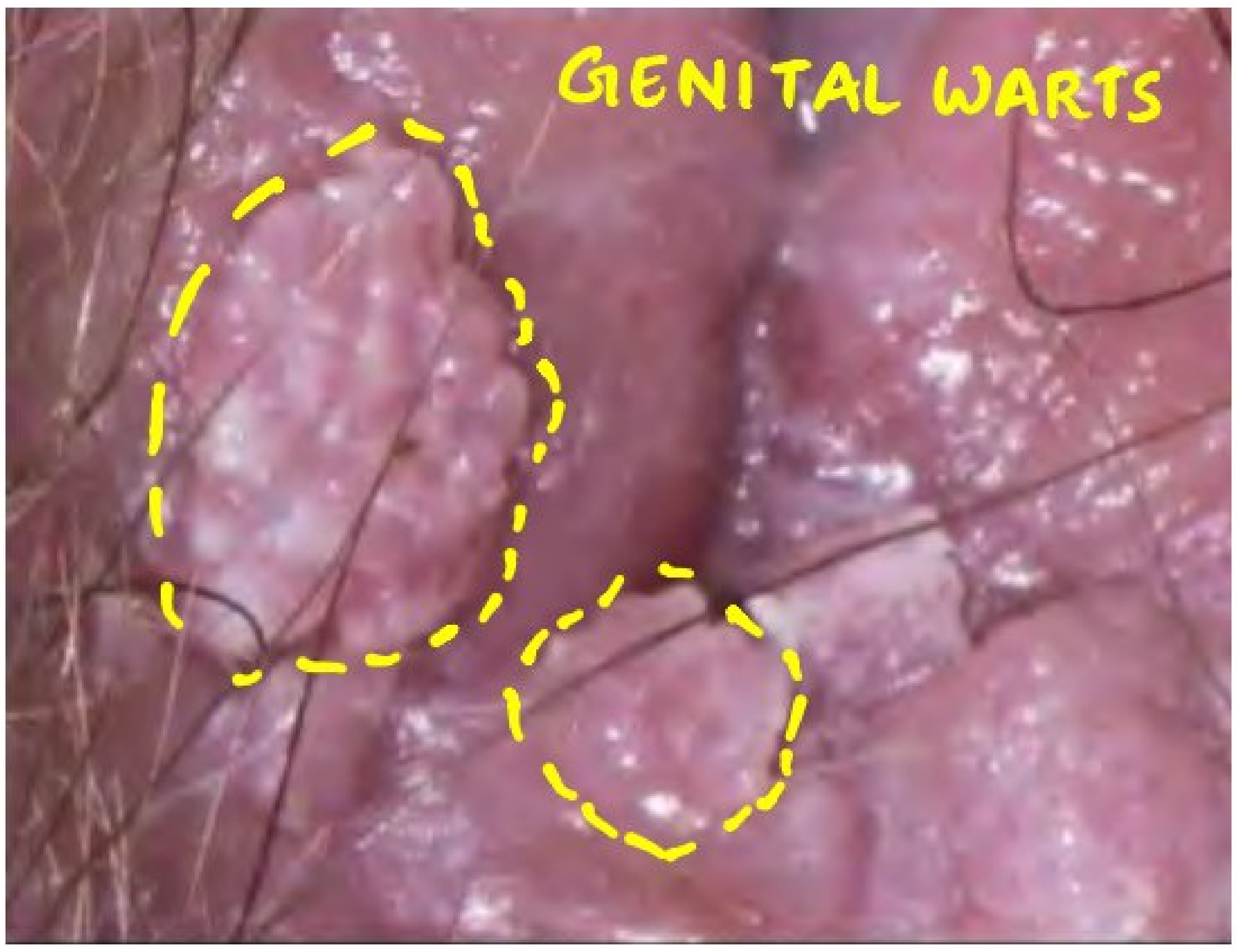
  ⊗ ↓ p53 ↳ hence cell IMMORTAL ○ → Complete Cell cycle ○○○○○○

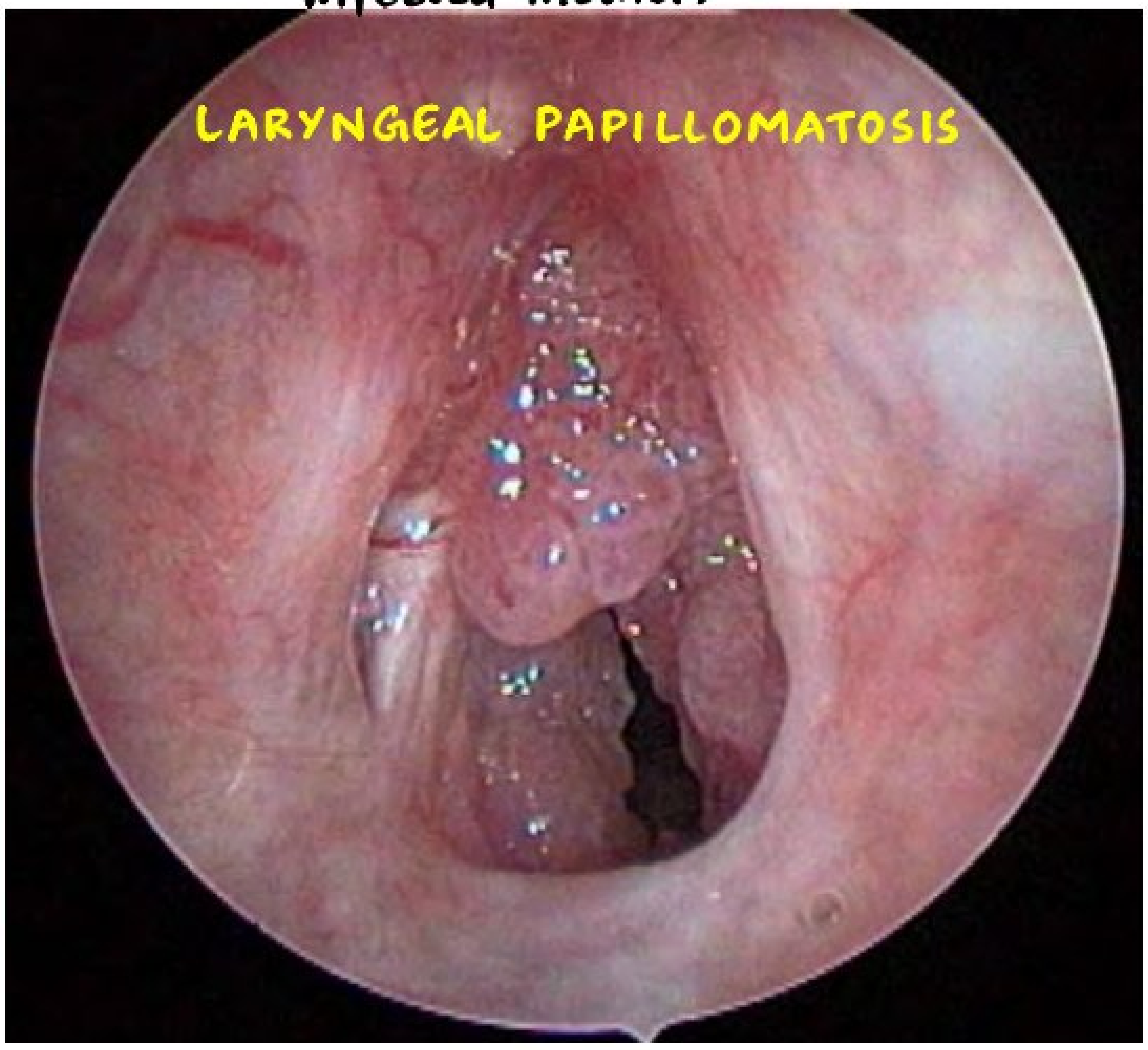
- Malignant Transformation : $E_6 + E_7$ ($E_7 > E_6$)

On HPE : KOILOCYTOSIS (perinuclear Halo)

LOW RISK TYPES : 6, 11 - cause GENITAL WARTS - CONDYLOMA ACUMINATA

↳ LARYNGEAL PAPILLOMATOSIS (Newborn Baby born to Infected mother)





CO-TEST : HPV + PAP • Sometimes PAP smear $\xrightarrow{f/u}$ HPV Triage (REFLEX TEST)

• HPV Infect → Squamous Epithelium → Basal Epithelial cells

• CA CERVIX - Vaccine preventable (Vaccine → Virus like particles → CAPSID Protein)
Virus

1. CERVARIX : BIVALENT (16,18) - 0,1,6m

2. GARDASIL : QUARIVALENT (16,18,6,11) - 0,2,6m → 70%

3. GARDASIL-9 : NONAVALENT (16,18,6,11,31,33,45,52,58)

FDA Approved: Ideal Age - 11-12yrs (can be given as early as 9yrs upto 45yrs)

0.5 ml i.m • if < 15yrs - 2 doses - 0,6m • if > 15yrs - 3 doses - 0,2,6m

• Syncopal Attack - Observation Time : 15 min • No Need for BOOSTER DOSE

• C/I Pregnancy. Screening has to be done even after giving vaccine.

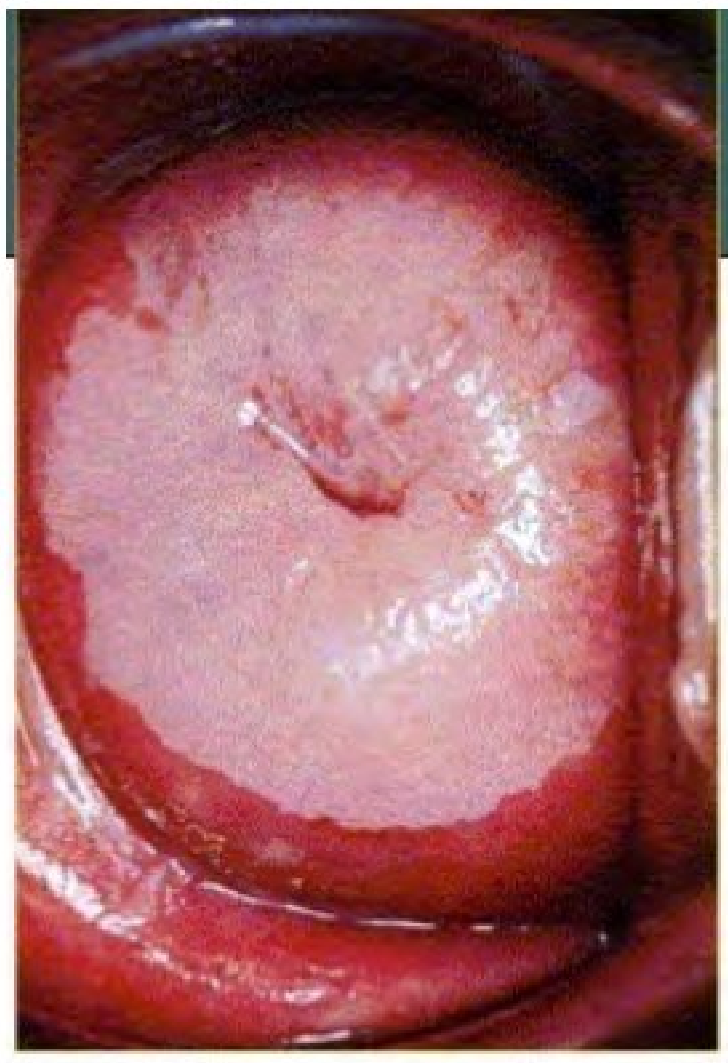
• VIA - Visual Inspection c̄ Acetic Acid 5% → if WHITE → AbⓃ areas - DYSPLASIA

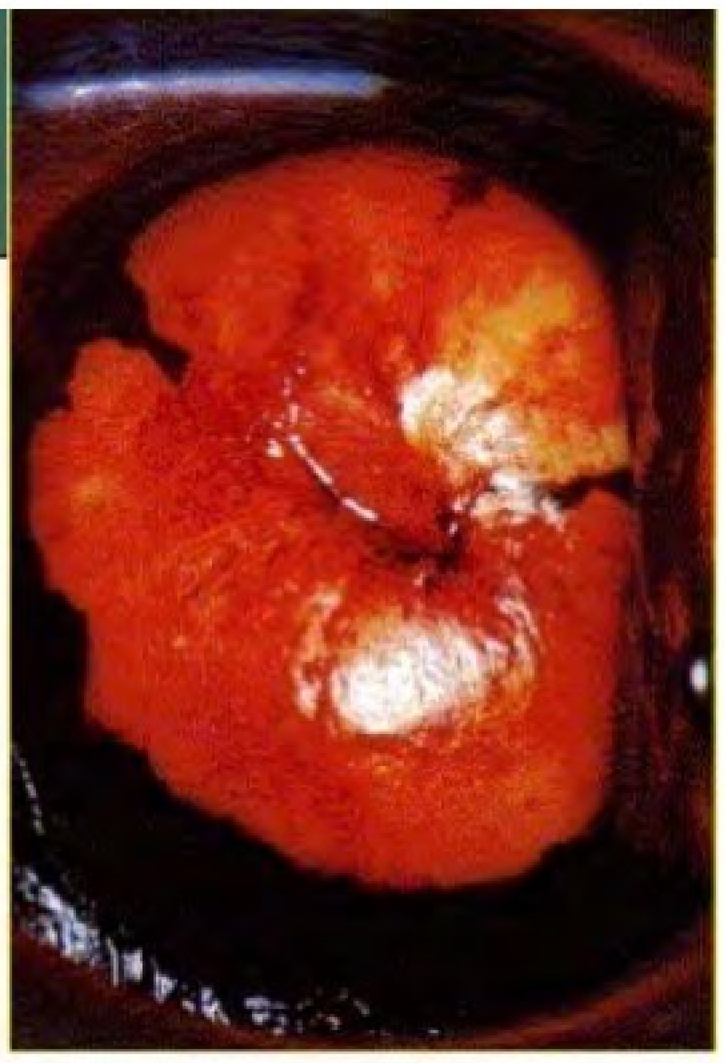
• VILI - Visual Inspection c̄ LUGOL'S IODINE (3-5%). Schillers Test

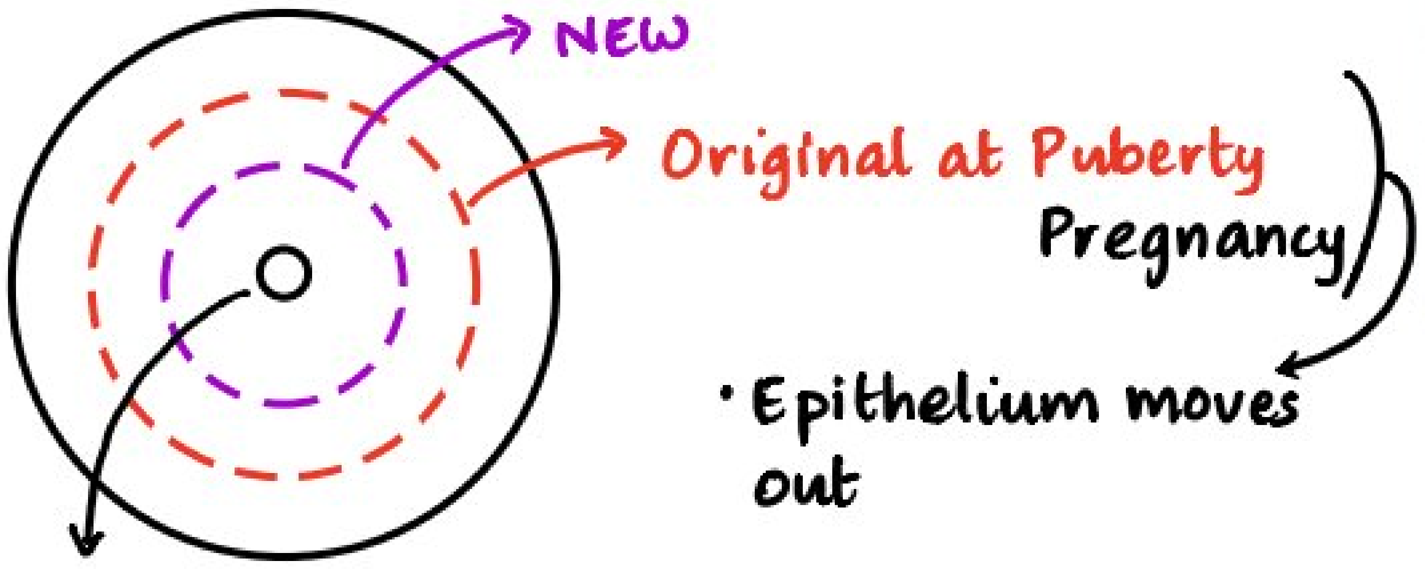
Ⓝ - Brown ; AbⓃ - Unstained areas → DYSPLASIA

• PAPS - Screening - CYTOLOGY Report

Transformation Zone
- Endo Cervix - Columnar } METAPLASIA
- Ecto Cervix - Squamous }
  - NOT Premalignant (Physiological)
  - DYSPLASTIC


VIA-positive


VILI-positive



• Eversion / Ectropion - RED Area bleeds on touch - Taken periovulatory

• NO C/I to take PAP smear. • Smear the spatula on Glass slide

• Relative C/I → Actively Bleeding • No Air drying → directly into FIXATIVE [95% ETHYL ALCOHOL + 5% ETHER]

- PAP Smear : BETHSEDA Classification

- Reactive and Reparative changes : healing inflammatory changes

L-SIL : Low grade Squamous Intraepithelial Lesion - Basement membrane - INTACT

ASCUS - Atypical Sq. Cell Undetermined Significance
AGUS - Atypical Glandular Cells Undetermined Significance

H-SIL : High Grade Squamous Intraepithelial Lesion

AbⓃ PAPS ⟶ Confirmation by Biopsy (Colposcope directed)

Microscope - Ectocervix
↓
Focal length : 30cm , Maximum magnification - 30x

ABNORMAL AREAS → Biopsy should be taken

1. Irregular Surface Contour
2. Pale mucosa
3. Acetic Acid - WHITE → Biopsy
   ↓
   GREY Areas - Metaplasia (no need for Biopsy)
4. Green filter in Colposcope
   - vessels can be seen easily
   - AbⓃ vascular pattern - RETICULAR vessels / PUNCTATE / MOSAIQ

- Colposcope Report : CIN-1, CIN-2, CIN-3 (Cervical Intraepithelial Neoplasia)

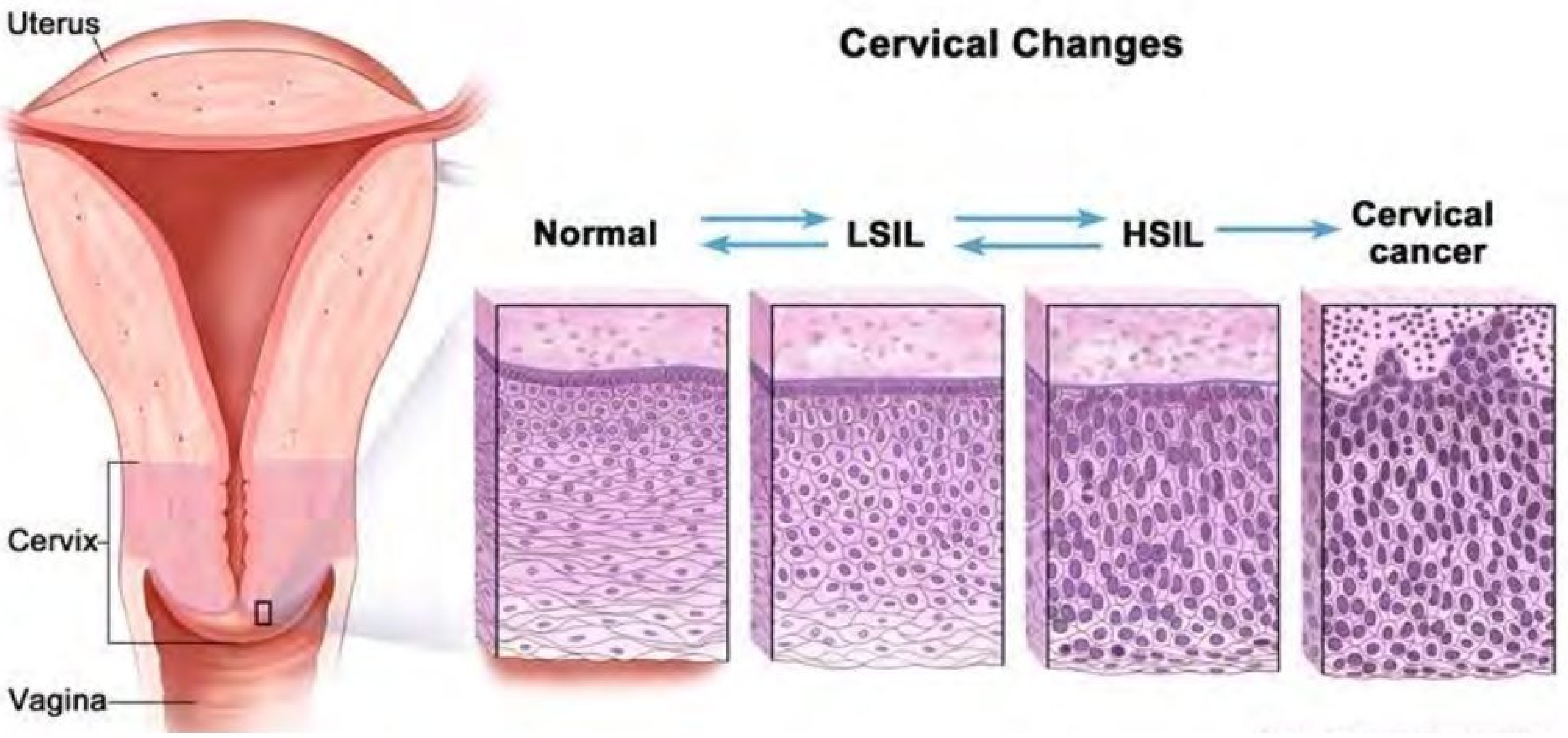
CIN-1 : MILD DYSPLASIA - Dysplasia in lower 1/3rd epithelium

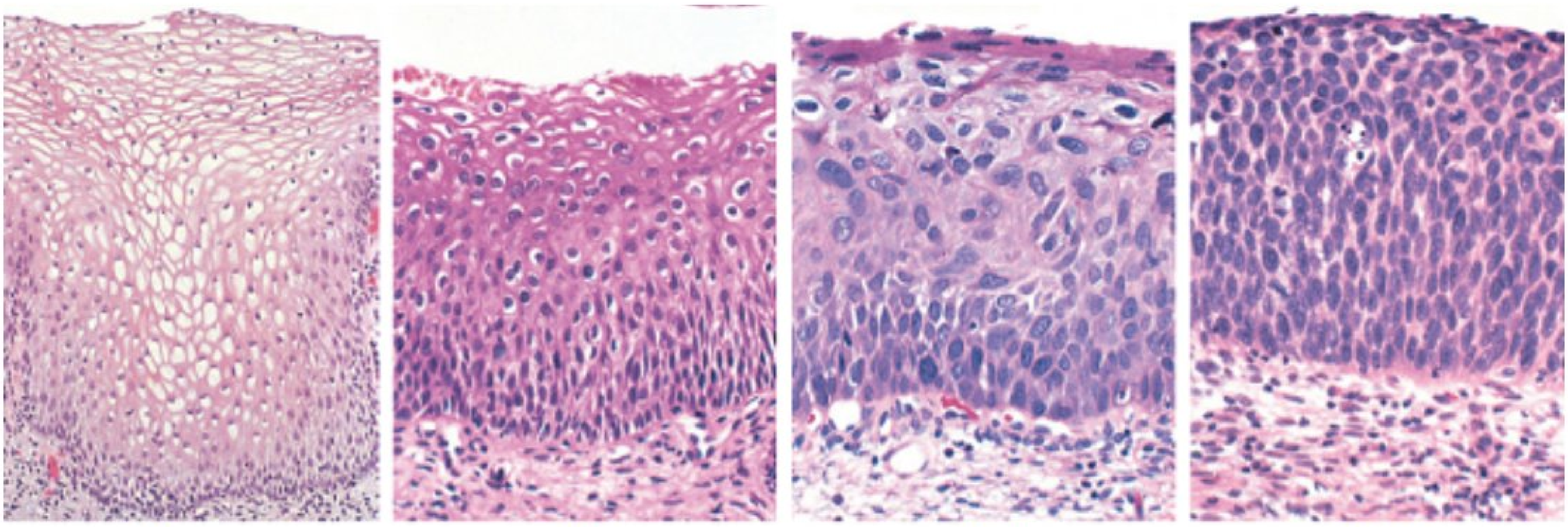
CIN-2 : MODERATE DYSPLASIA in lower 2/3rd epithelium

CIN-3 : SEVERE DYSPLASIA in lower > 2/3rd epithelium

CIN-1 : L-SIL

CIN-2,3 : H-SIL





Normal CIN I CIN II CIN III

Management of Pre-Invasive Lesion → Excision > Ablation

↳ Better because Tissue Sample Sent (Tz)

Confirmed CIN-1 $\xrightarrow{1\%}$ Carcinoma Risk

↓

Conservative Mx: CO TEST (PAPs + HPV) - 12 monthly for 2 years

↓ if CIN-1 Persistent - then Treat

↳ Average time taken by CIN-1 to regress

- CIN-2 → 5% Cancer Risk
  CIN-3 → 12-40% Cancer Risk

↳ Treatment: 1. ABLATION (CRYOABLATION) → OPD Procedure; No Bleeding. Long Term S/E: Persistent Watery Discharge

↳ CO_2 / Nitrous oxide → Crystallization of Intracellular Water → Dessication of Cell → Cell death

2. EXCISIONAL: ① LEEP = LLETz Loop Electro surgical Excisional Procedure
Large Loop Excision of Transformation Zone

- OPD Procedure
- Less time (~20 min) required
- NO Major Bleeding
- Sample from Tz

- if Abnormal PAPs → directly Rx s̄out confirmation
  ↓
  Do LEEP

② CONIZATION

- excisional
- Larger chunk of tissue
- done ↓ OT

- Samples from - i) Ectocervix
  ii) Endocervix
  iii) Transformation zone (Tz)
  iv) Junctional zone
  ↳ To see basement memb⊕/not

INDICATIONS :

1. Tz cannot be completely seen (unsatisfactory colposcopy)
2. Discrepancy b/w Cytology and Histopathology
3. ECC - Endocervical Curettage. ⊕ve Dysplastic cells in ENDOCERVIX
4. Suspicion of INVASION.

Q. PAPs Report - L-SIL. Next Step? Ans: Colpobiopsy

Q. PAPs: H-SIL. Next Step? Ans: Colpobiopsy + ECC (Mandatory)

PAPs → ASC-US → Next step? → HPV Triage / Reflex —f/u→ c̄ HPV Test

→ ⊖ve - do CoTest after 3yrs

→ ⊕ve - Colpobiopsy

- ASC-H ⟶ do Colpobiopsy + ECC
- AGUS ⟶ ECC + Biopsy

Q. 40yr old P_2L_2. CIN-2 on Colpobiopsy. Next Step? Ans: LEEP

RISK FACTORS for CERVICAL CANCER

1. Early age at 1st Intercourse
2. Multiple Sexual Partners
3. Multiparity
4. Low Socio-economic status
5. STD
6. HIV
7. Pre-Invasive Lesions
8. OCP - can ↑ Risk; only if used consecutively ≥ 5yrs
9. Smoking - R/F for Squamous Cancer

(MC) Histology - 69% → Squamous ⟶ Large Cell Non-keratinizing histology

25% → Adeno

- BIMODAL AGE Distribution : 1st Peak - 3rd and 4th decade

  2nd Peak - 5th and 6th decade

- SPREAD - Direct spread / Lymphatic spread
- (MC) Presentation : Irregular Bleeding
- Most specific : POST COITAL Bleeding (PCB)

s/i ① Cervical cancer ② Ectropion ③ Cervical polyp ④ Cervicitis ⑤ Prolapse

do Clinical examination + PAPs

- PCB → visible growth or cervix ⟶ PUNCH Biopsy

CAUSE OF DEATH: ⓂⒸ cause UREMIA - d/t Renal failure

- ⓂⒸ site for DISTANT METS - LUNGS
- OVARIAN Spread - Risk < 0.5% (so need for OOPHERECTOMY)

- PROGNOSIS: depends on Stage > LN Status

FIGO STAGING: MRI - Best modality for Parametrial Invasion

LN status - CT PET > CT Guided Biopsy

CLINICAL:

STAGE 1: Strictly confined to cervix

1A
- $1A_1$ - < 3mm in depth
- $1A_2$ - ≥ 3mm and < 5mm in depth

1B
- $1B_1$ - ≥ 5mm depth of invasion and < 2cm in greatest dimension
- $1B_2$ - ≥ 2cm , < 4cm in greatest dimension
- $1B_3$ - ≥ 4cm - maximum dimension

STAGE 2: 2A - upper 2/3rd of vagina spread s̲out invasion of Parametrium
- A_1 < 4cm
- A_2 ≥ 4cm

2B - Cancer spread 2/3rd upper vagina c̲ parametrium

STAGE 3:
- 3A - cancer involving lower 1/3rd vagina
- 3B - Spread to Lateral pelvic wall
- 3C - C_1 → involvement of pelvic LN
  C_2 → Para aortic LN

STAGE 4:
- 4A - involvement of Bladder and/or Rectum
- 4B - Any distant spread (eg: Inguinal spread)

TREATMENT : ≥ 4cm or beyond Cervix (from 2B onwards) $1B_3$, $2A_2$ } Primary Rx: CHEMORADIATION

: for all other Primary Rx : SURGERY

- For $1A_1$: < 3mm in depth - Surgery done - CONIZATION. If family is Complete, can also do hysterectomy. Type 1A - Simple Extrafascial Hysterectomy
- For $1A_2$: TYPE 2B - Modified Radical Hysterectomy = WERTHEIM'S HYSTERECTOMY + PELVIC LYMPHECTOMY
- For $1B_1$, $1B_2$, $2A_1$ } TYPE 3 (c) Radical = MEIG's HYSTERECTOMY + PELVIC LYMPHADENECTOMY

- **For Type 2 :** Uterus, cervix, $1/3^{rd}$ vagina, $1/2$ Parametrium, clamps on Uterosacral and cardinal ligaments (midway b/w origin and insertion). Uterine artery ligated after it crosses the ureter

- **For Type 3 :** Uterine Cervix, upper $2/3^{rd}$ vagina, all parametrium. Clamps on Uterosacral and cardinal ligament. Uterine artery ligated at its origin

CHEMORADIATION

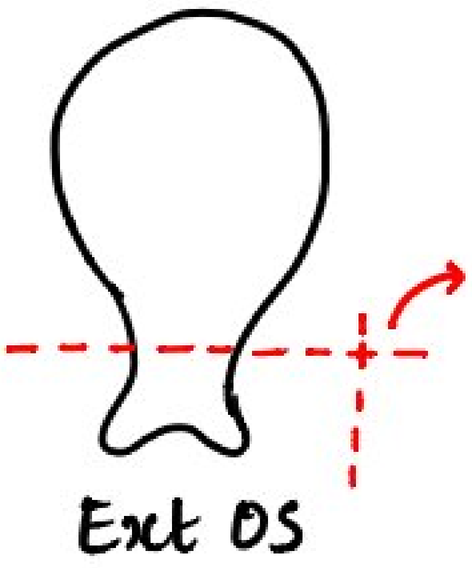
- concurrent Chemo Rx + Radio Rx
- chemo → CISPLATIN > 5 FU
- Radio → 1. EBRT (External Beam Radiotherapy) Then 2. BRACHYTHERAPY (Intracavitary)

(MC) used in Gynaec malignancy
aka TELETHERAPY

- we use Cesium
- Pelvis - 50Gy over 5wks

IRIDIUM

- HDR Therapy (High Dose) → at > 12 Gy/hr
- LDR Therapy (Low Dose) → at < 2 Gy/hr



POINT 'A' : 2cm above Ext. OS and 2cm lateral to cervix
↳ corresponds to ureter (Total Dose : 80-90 Gy ~ 8000 cGy)

OVARIAN CANCER

- NON SPECIFIC Signs and Symptoms
- NO Pre-Invasive Stage
- NO Universal Staging

ADNEXAL MASS : Benign or Malignant

BENIGN	MALIGNANT
• Reproductive Age • Pain ⊕	• Extremes of age (adolescent, post menopausal women)
	• painless to begin with
O/E : U/L ; cystic consistency Tender as well	O/E : B/L ; Variable consistency, Non-Tender
IOC : TVS • Anechoic, Unilocular eg: Follicular cyst (MC) cyst of Ovary	IOC : TVS • Solid, Thick septae, High vascularity, papillary excrescences
• Follow up on USG	• a/w ASCITES → Enlarged LN, Matted Bowel loops need to operate.

- Unilocular anechoic mass → do Tumor marker → Old age : CA 125
  - → Adolescent : ① HCG ② AFP

Q. Adnexal mass in Pregnancy s/i 1st TM. What next ?

Ans: Do USG → possibly Corpus luteum (will regress by 12wks) → So, Repeat USG in 2nd TM.

Q. If Adnexal mass in Pregnancy s/i 2nd TM. What next?

Ans: if HIGH Risk features ⊕ } Operate in 2nd TM (Risks are minimum)
>10cm → can undergo TORSION / RUPTURE }

RISK FACTORS OF OVARIAN CANCER

1. ↑ Estrogen ~ Epithelial proliferation
2. ↑ No. of Menstrual cycles ~ ↑ ovulation
3. Early menarche ; Late menopause
4. Obesity
5. Nulliparity
6. PCOS
7. BRCA - 1, 2

Smoking } NOT A R/F
HRT }

PROTECTIVE FACTORS

1. OCP's
2. Breast feeding
3. Anovulation
4. Tubal ligation and Salpingectomy
5. Hysterectomy

OVARIAN / TUBAL / PERITONEAL Carcinomatosis : Origin from Fallopian Tube

EPITHELIAL CELL TUMORS	GERM CELL	SEX CORD- STROMAL
• (MC) - 90% 1. SEROUS - (MC) 75% 2. ENDOMETROID - 10% 3. MUCINOUS - 10% 4. BRENNER'S 5. CLEAR CELL	• 5-8% • MATURE CYSTIC TERATOMA aka DERMOID CYST • (MC) Germ cell cancer - Immature Teratoma f/b DYSGERMINOMA • ENDODERMAL SINUS TUMOR (EST) • YOLK SAC Tumor • CHORIOCARCINOMA	• 3% • GRANULOSA • SERTOLI - LEYDIG • THECOMA } STROMAL TUMORS • FIBROMAS } STROMAL TUMORS

- (MC) Tumor - SEROUS CYSTADENOMA
- (MC) CA - CYST ADENO CARCINOMA

GENERAL:

1. SEROUS :
- s/i older women ; 6th and 7th decade (Peak - 60yrs)
- B/L
- Non-specific Signs and symptoms (IBS)
- Diagnosis : At Late Stages → BAD Prognosis [Out of the 3 - WORST Pregnancy]
  HIGH Mortality Rate

2. GERM CELL :
- s/i Younger women (10-30yrs) ; peak 2nd decade , U/L
- Non specific signs and symptoms ; also presents c̄ precocious puberty
- They secrete HCG
- Diagnosed in Early Stage - BETTER Prognosis

3. SEX CORD - STROMAL
- occurs at any age (peak at Perimenopausal women)
- U/L
- Non-specific ; AUB ; Virilization
- Diagnosed Early Stage : BEST Prognosis

EPITHELIAL - MUCINOUS VARIETY

- U/L
- a decade earlier
- CEA / CA 19-9
- Grows to Huge Sizes (20cm)
- Early Stage diagnosis
- BETTER Prognosis compared to SEROUS
- PSEUDOMYXOMA PERITONEI : loculated mucinous collection
- ENDOMETROID OVARIAN TUMOR :
  - very high / highest risk of co-existent Endometrial cancer.
  - Pt can develop ENDOMETRIOSIS → (MC) c̄ CLEAR CELL

- EPITHELIAL

90% SPORADIC
10% FAMILIAL → occur decade earlier
↳ a/w Genetic mutation

Lifetime RISK
- In BRCA-1 → upto 45%
- In BRCA-2 → upto 25%
- HNPCC [LYNCH] → upto 15%

- 1st degree Relative Familial cancer : ↑ses Risk by 3 times
- K/c/o BRCA 1/2 Mutations → Prophylactic BSO → 35-40yr once family is Complete
  ↳ Reduce Risk of Breast CA by 50%
- If Family h/o CA ; no mutations - in them do screening → ① TVS
  ↳ ② CA-125

- Start screening at 35 yrs age ; do annually

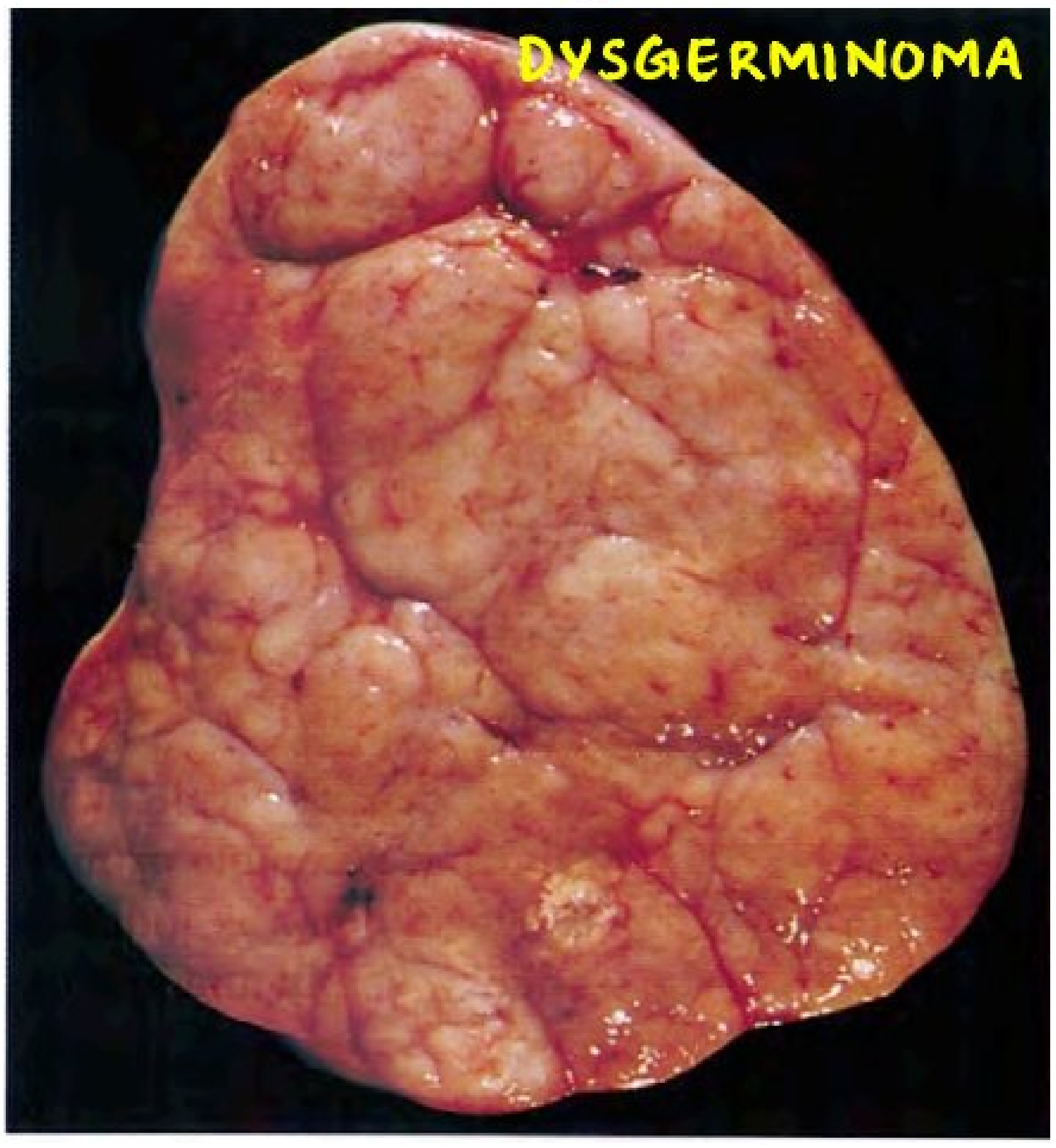
DYSGERMINOMA : Solid, fleshy, Lobulated, tan. U/L, but can be B/L in 15% case (Highest Risk of B/L)

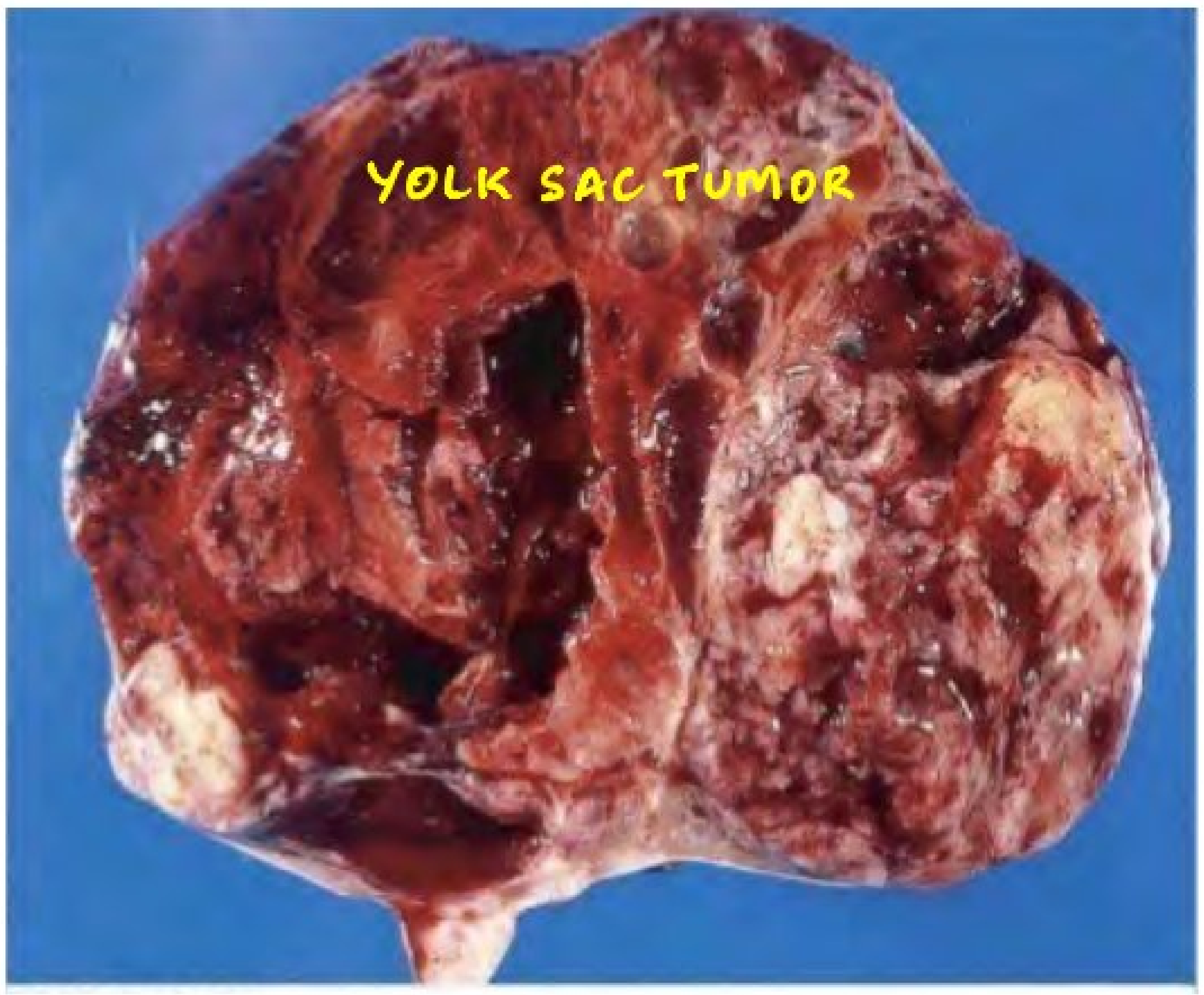
- Tumor marker : LDH (also secrete HCG/PLAP)
  if AFP ⓧ → BEST Prognosis

- Only Ovarian tumor which is RADIOSENSITIVE.

EST (YST) : secrete AFP (main), LDH. Never HCG

- WORST Prognosis → because very rapidly growing tumor → A/c Abdomen Presentation





DERMOID [Mature Cystic Teratoma]

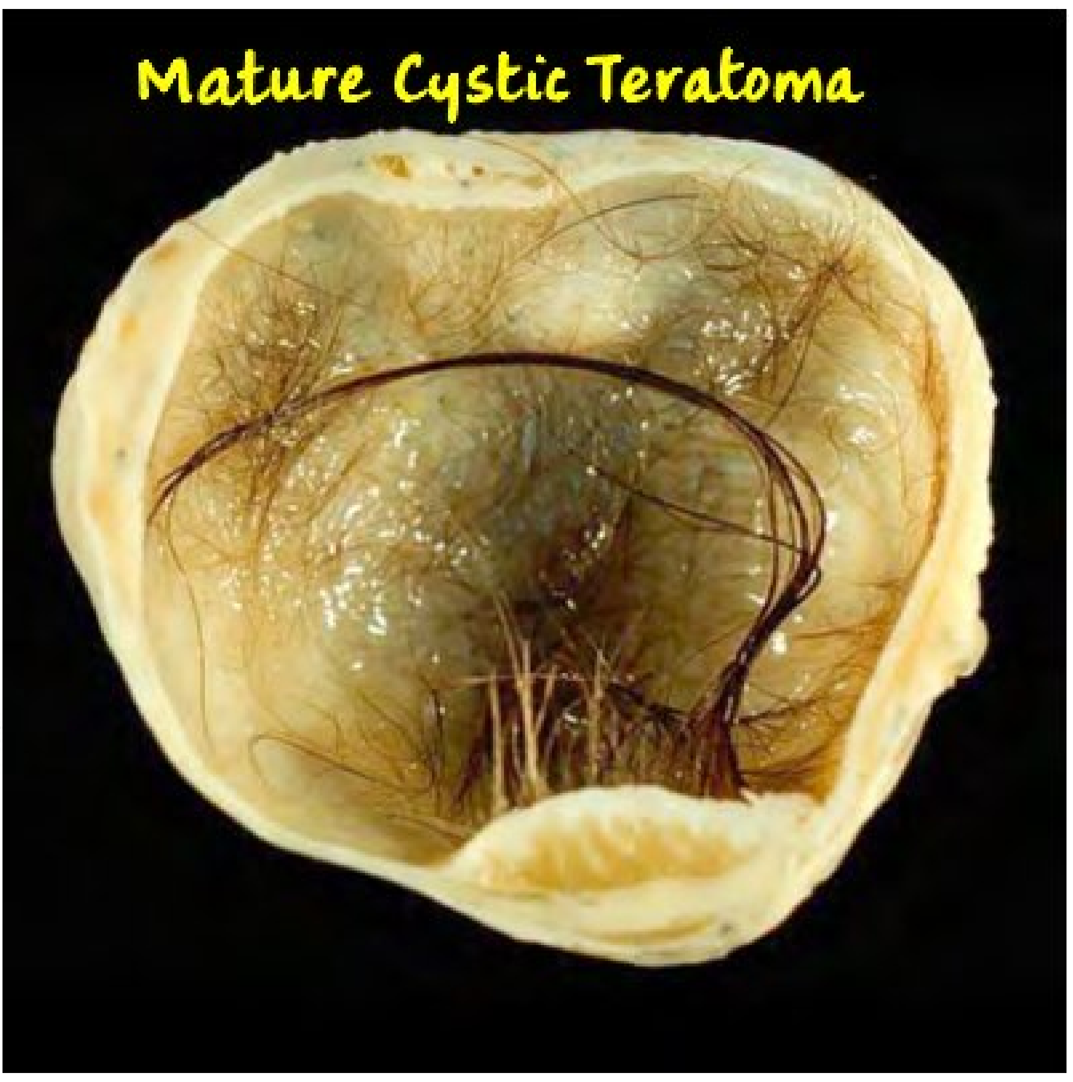
- (MC) Ovarian tumor in Reproductive Age grp - DERMOID
- (MC) " in Pregnancy - DERMOID
- (MC) " to undergo TORSION

On USG : ROKITANSKY PROTRUBERANCE → Site: Malignancy

- Tip of ICEBERG

(MC) in Dermoid - Squamous Cell Cancer

- BENIGN ; Risk of Malignancy (0.2 - 2%)
- U/L ; can be B/L upto 10%



SEX CORD STROMAL TUMORS

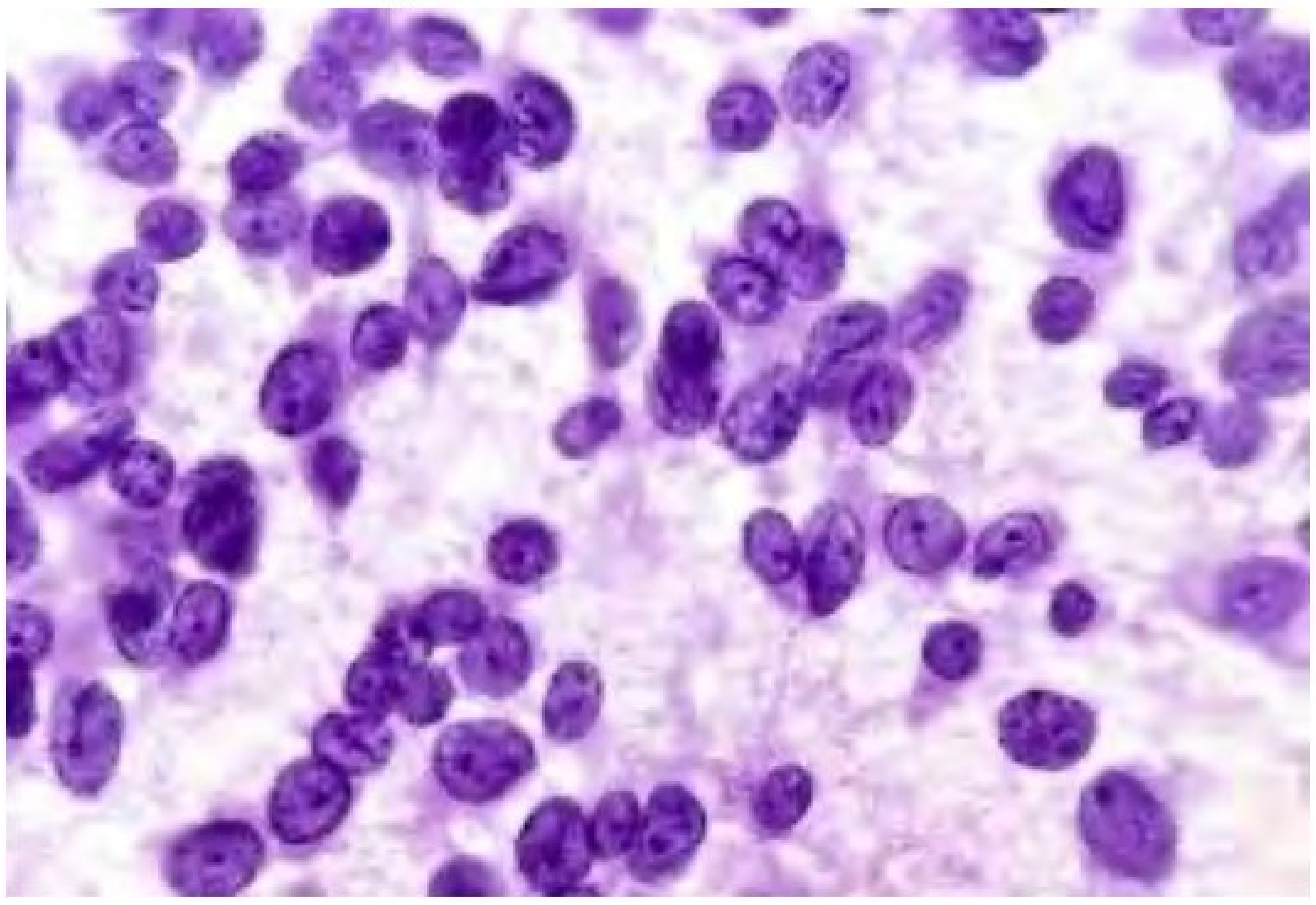
GRANULOSA CELL TUMORS : Estrogen ; Tumor Marker - INHIBIN ; peak : Perimenopausal AUB

- also ↑ Risk of ENDOMETRIAL CANCER

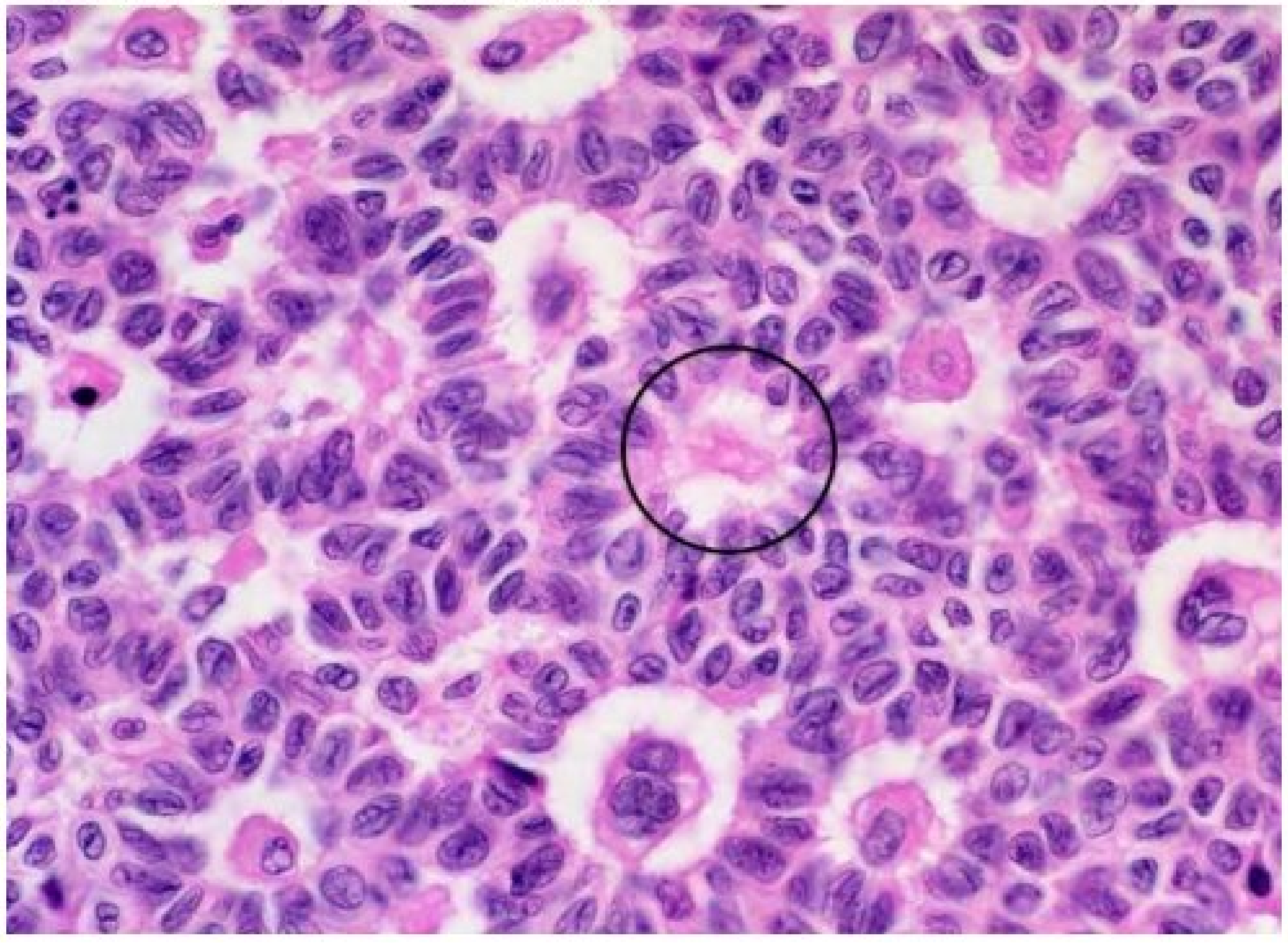
On HPE : CALL EXNER Bodies ; Coffee Bean Nucleus (a/s/i BRENNER's) → Benign Solid

↳ On HPE : WALLTHARD CELL NEST

Coffee Bean Nucleus



CALL EXNER Bodies



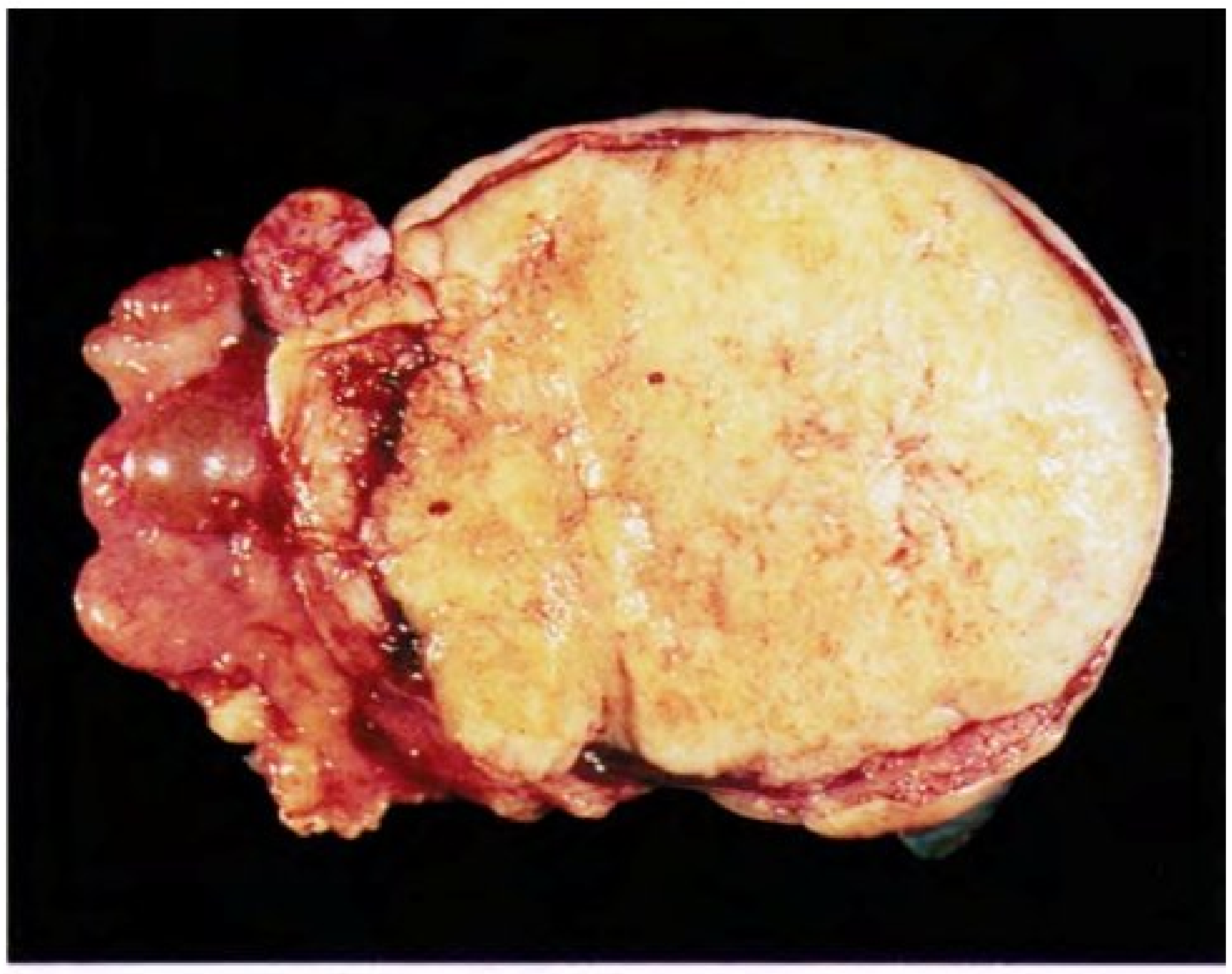
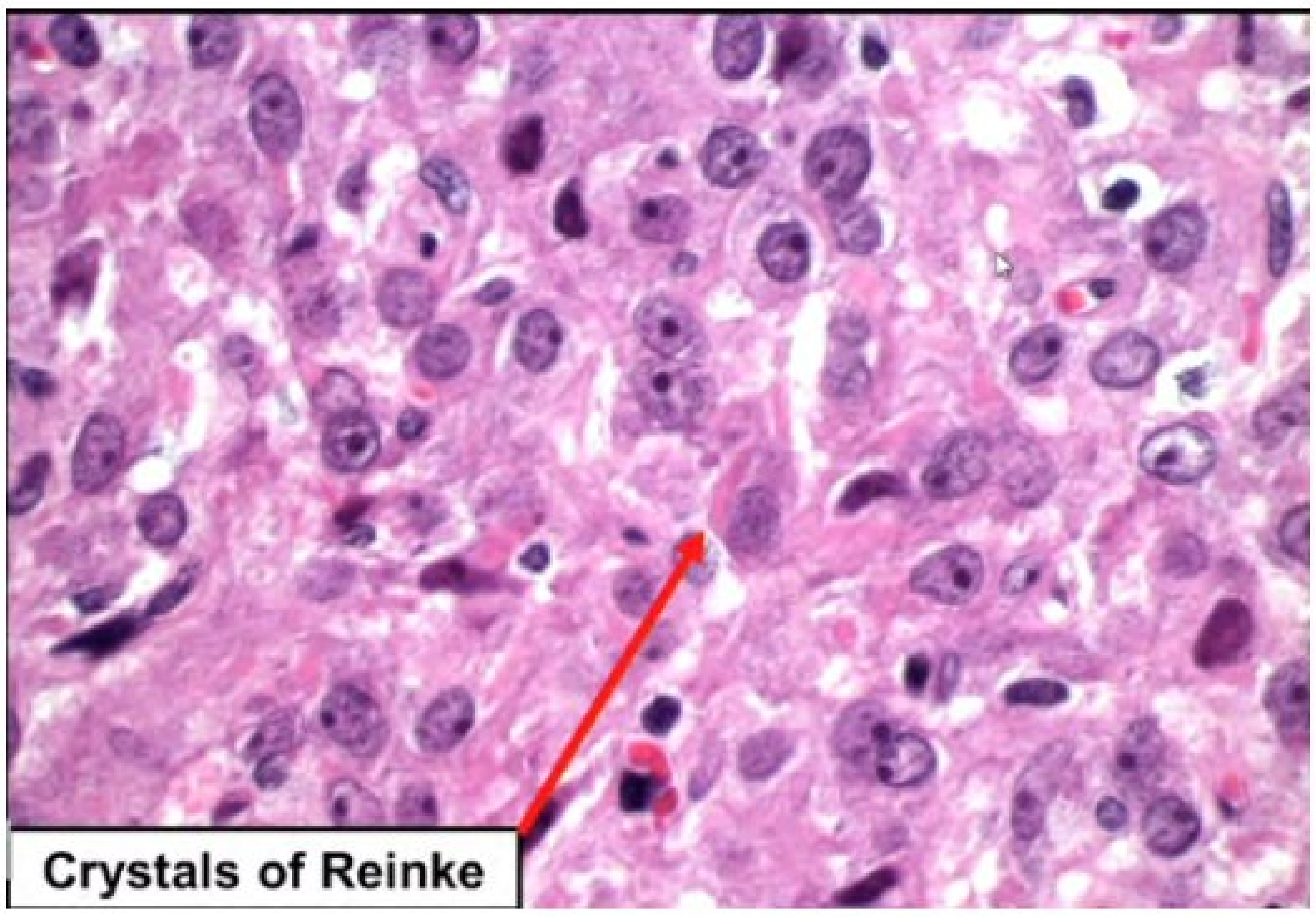
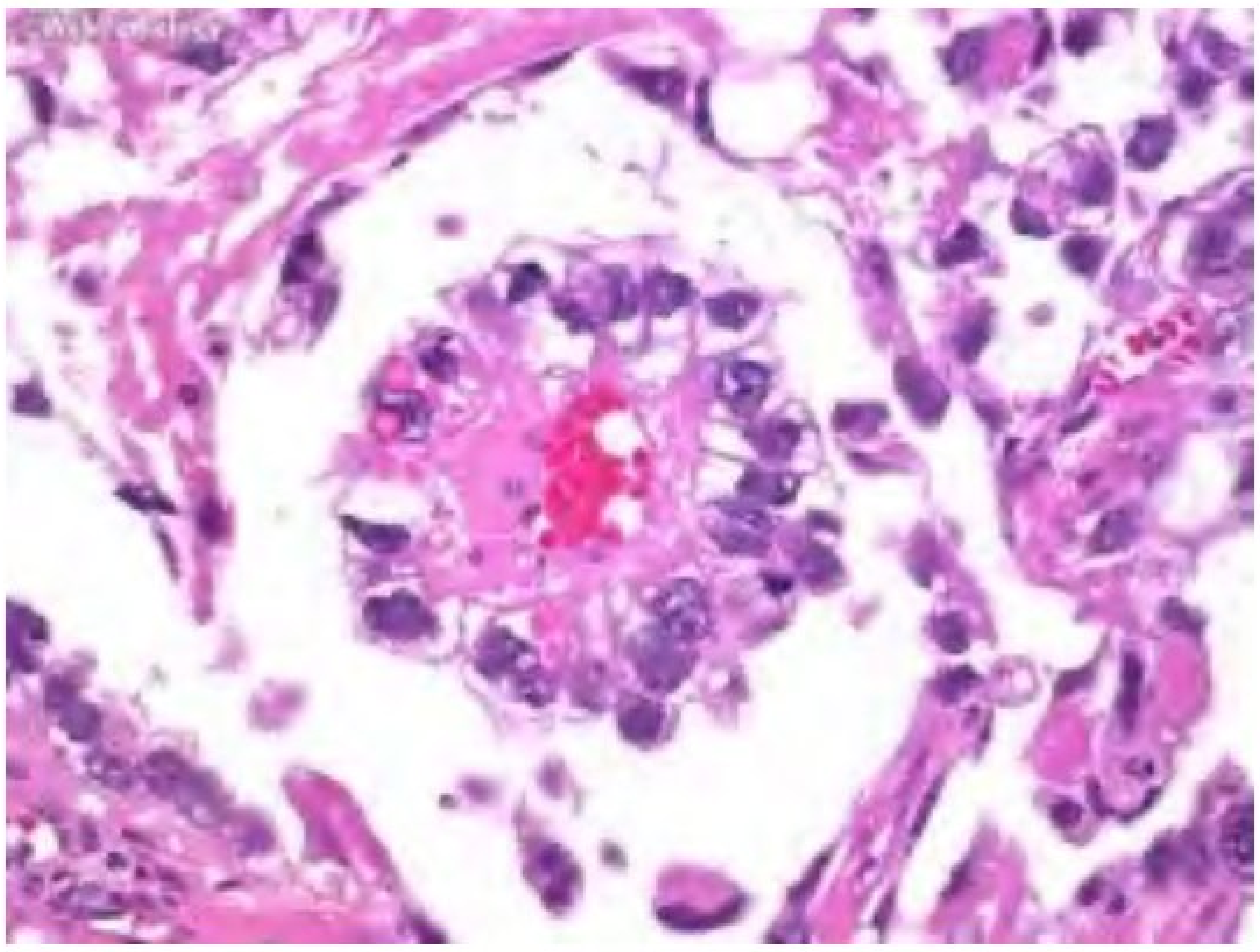


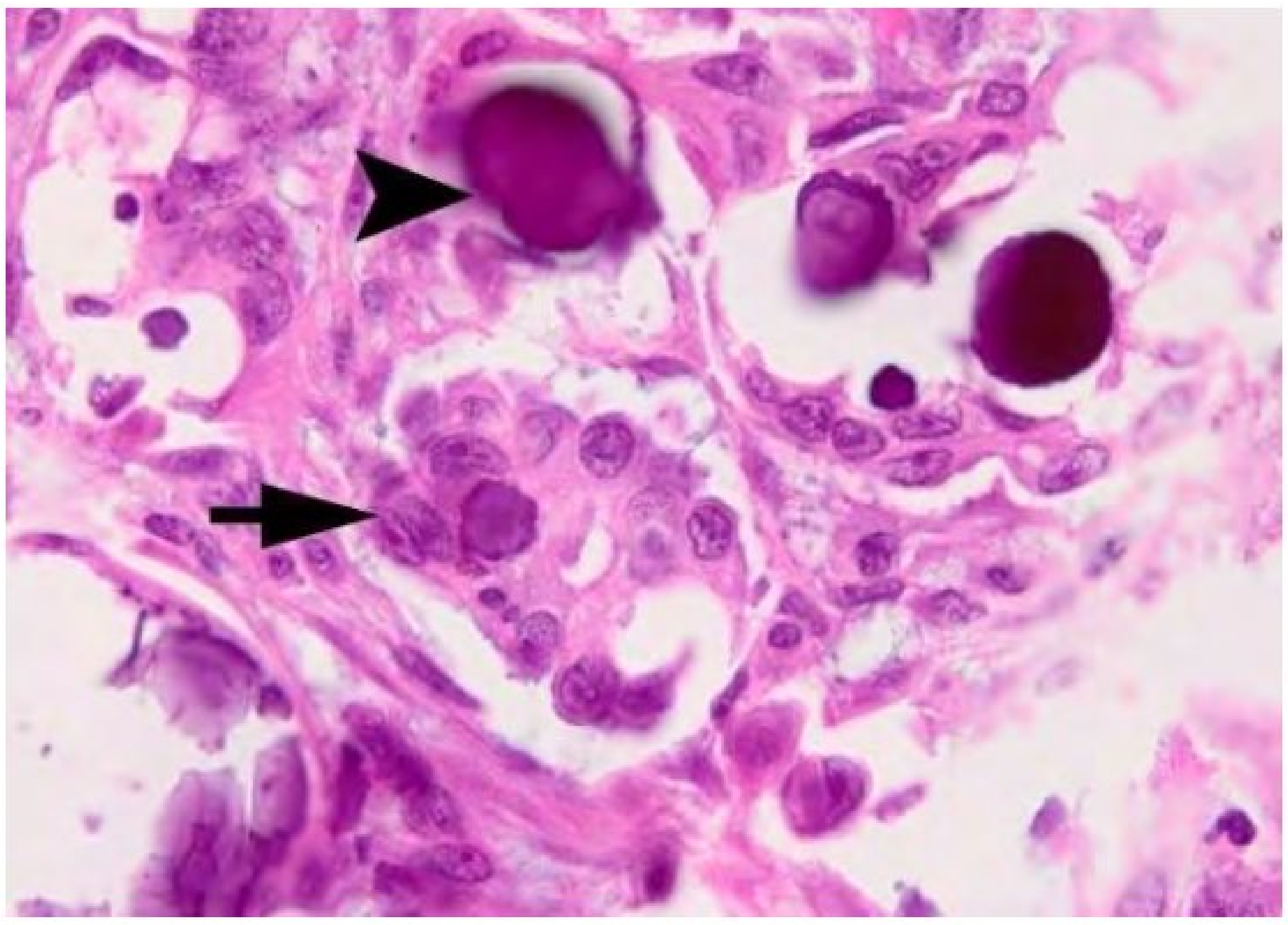
Figure 9-2
ADULT GRANULOSA CELL TUMOR
The sectioned surface is uniformly solid and yellow.



REINKE'S CRYSTAL in LEYDIG CELL TUMOR



SCHILLER DUVAL BODIES in EST



PSAMMOMA BODIES in SEROUS

FIBROMA

- Stromal tumor
- SOLID Ovarian tumor
- Benign
- MEIGS Synd
  - Stromal tumor
  - Ascites
  - Pleural Effusion
- Ascites and Pleural Effusion - Spontaneous regression

PSEUDOMEIGS : Non Stromal tumor + Ascites + Pleural Effusion

- SIGNET RING Cell : KRUKENBERG TUMOR OF OVARY → 2° in Ovary
  - B/L (80%), can be U/L
  - capsule can be intact
  - shape of ovary maintained

(MC) 1° - Stomach (Pylorus)

- Retrograde lymphatics

STAGING OF OVARIAN TUMOR [FIGO] - done on basis of Surgery

(MC) Route tumor Exfoliation

STAGE 1:

A - U/L Ovary B - B/L Ovaries

C - 1. Intra operative Capsule Rupture

2. Pre-Op Capsule Rupture

3. Pressure of Malignant Ascites

STAGE 2: A - Spread to uterus and/or FT

B - spread to other pelvic structure

STAGE 3 : A → A_1 - ⊕ Retroperitoneal LN

→ A_2 - Microscopic Extrapelvic Peritoneal Spread

B - Macroscopic < 2cm. Extrapelvic Peritoneal Spread

C - Macro > 2cm. Extrapelvic Peritoneal Spread or Involvement of capsule of Liver/Spleen.

STAGE 4 : A - Malignant Pleural Effusion

B - Distant spread / inguinal LN; parenchyma of Abdominal organs.

Stage 1 and 2 - Early Stage CA

Stage 3 and 4 - Late Stage CA (Advanced)

STAGING LAPAROTOMY

1. Midline Vertical Incision
2. If presence of ASCITIC fluid : Send for cytological study
3. Inspection and Palpation of all abdominal organs
4. Random Peritoneal Biopsy
5. TAH + BSO → Type 1 Hysterectomy
6. Infracolic Omentectomy
7. Pelvic and Para aortic LN Sampling
8. Closure

U/L Salpingo opherectomy - fertility preserving Surgery for Ovarian Cancer

- In Case of MUCINOUS Ovarian Cancer - add appendicectomy
- K/c/o Ovarian cancer : Pre-Op CT Scan done - Not used for Staging

POST-OP TREATMENT

EPITHELIAL

- all stages except Stage 1A and Stage 1B Grade 1 need Chemo Rx
- PACLITAXEL + CARBOPLATIN

(DOCETAXEL / CISPLATIN)
6 cycles of Chemo

GCT

- all Stages except DYSGERMINOMA Stage 1 needs Chemo

SEX CORD

- all advanced stages need chemo

BEP x 6 cycles
- Bleomycin
- Cisplatin
- Etoposide

ENDOMETRIAL CA

- Risk factor
- ↑ Estrogen (excess/unopposed)
- ↑ menstrual cycles

1. Early menarche
2. Late menopause
3. Infertility
4. Nulliparity
5. PCOS
6. BRCA-1, BRCA-2

   HNPCC → Highest Risk upto 70%
7. COWDEN's Syndrome
8. Obesity
9. Hypertension
10. DM

(8, 9, 10: } Endometrial CA CORPUS CANCER Syndrome)

11. Ovarian Tumor < Endometriod / Granulosa
12. TAMOXIFEN (SERM)
    - on long term administration
    - ↑ Risk
    - RALOXIFENE doesnot ↑ Risk
13. HRT - Ⓔ alone

- PROTECTIVE FACTORS

1. Smoking
2. OCP's (Long Term OCP's → THIN)
3. Breast feeding
4. Physical activity
5. Green Tea

- HISTOLOGY

TYPE - 1	TYPE - 2
• Endometroid (Grade 1 and 2) ; (MC) 80%	• Non-Endometroid, Papillary Serous Clear Cell

TYPE - 1	TYPE - 2
• Estrogen dependent • Pre-Invasive lesion in the form of HYPERPLASIA • GOOD PROGNOSIS • Obesity • a/w PTEN Mutation, KRAS (COWDEN'S SYNDROME) → Gate keeper gene for Endometrial cancer	• Non-Endometroid (Grade 3) • 20% • Not Estrogen dependent • NO Pre-invasive lesion • POOR PROGNOSIS • THIN Women • p53 [if a/w p53 - High Grade Tumor if a/w KRAS - Low Grade Tumor

• PRE-INVASE LESIONS - Endometrial Hyperplasia : Pathological Diagnosis. There are 4 Types

1. SIMPLE HYPERPLASIA s̄out ATYPIA : 1% Risk of CA - Proliferation of gland stroma aka CYSTIC GLAND HYPERPLASIA

2. COMPLEX HYPERPLASIA s̄out ATYPIA : 3% Risk of CA - Glands >> Stroma, crowding glands

3. SIMPLE WITH ATYPIA - 8% Risk of CA

4. COMPLEX WITH ATYPIA - 29% Risk of CA

• Hyperplasia s̄out Atypia : Treatment 1) Progesterone continuous - TOC
↓
decidualisation → Atrophy

2) MIRENA >> MPA (ORAL) for 6 months → after 6 months Repeat sampling (Endometrial)

• Hyperplasia c̄ ATYPIA : OPD Procedure - Report

1st confirm Malignancy or not ? → do D and C and Confirmation in O.T
↓
if again Report comes as HYPERPLASIA c̄ ATYPIA
TOC : HYSTERECTOMY - post menopause + BSO
↓
if refused 2nd line : Prog. MIRENA > ORAL (MEGESTEROL ACETATE)

Endometrial CA

• occurs in 5th - 7th decade (Peak - 60yrs) • (MC) Route of spread - DIRECT

• PRESENTATION : IRREGULAR Vaginal Bleeding - (MC) Presentation

- most specific → POST-MENOPAUSAL BLEEDING (PMB) → Only ≤ 10%

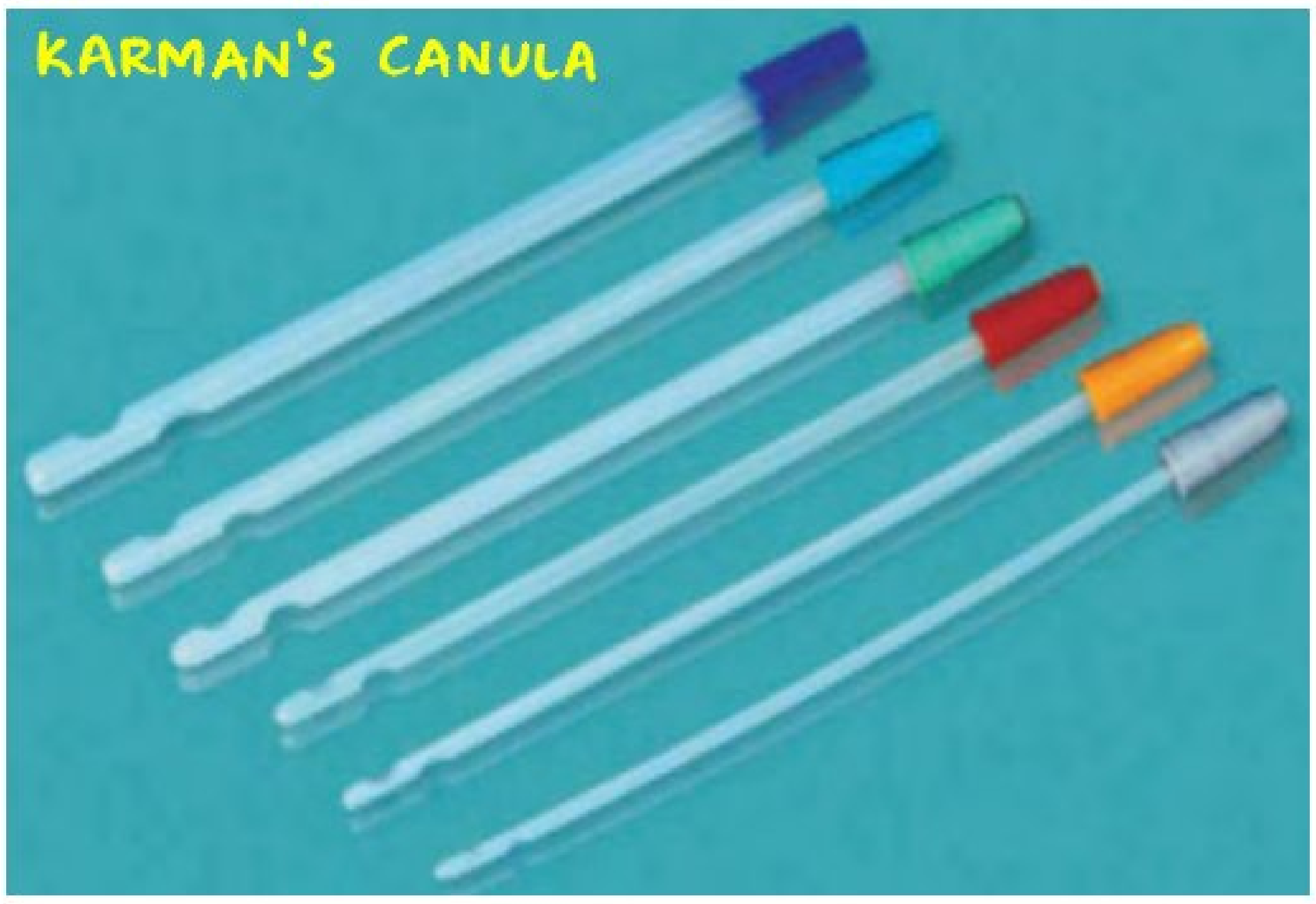
Other causes of PMB :

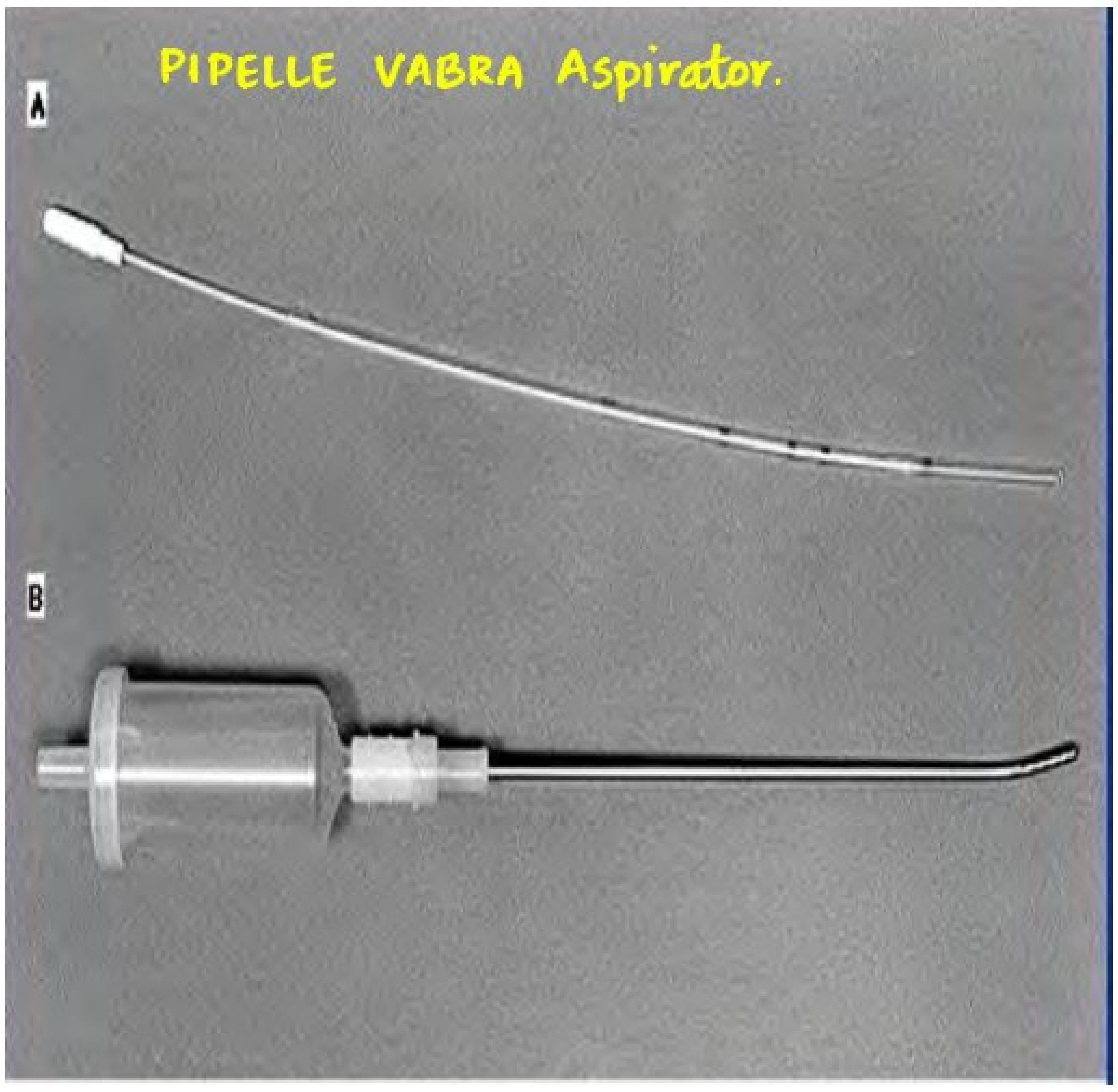
1. Atrophic Endometritis (Senile)
2. Endometrial polyp
3. Endometrial hyperplasia
4. Atrophic vaginitis
5. CA Cervix

(MC) cause
→ Pyometra : ENDOMETRIAL CA
→ Pyometra in INDIA : CA CERVIX
→ Post Menopausal Bleeding in INDIA : CA CERVIX

- 1st Investigation in PMB : TVS → Endometrial Thickness - FIGO ≥ 5mm → ↑ Risk of C·A
  ↘ ACOG ≥ 4mm → ↑ Risk of C·A

- IOC : Endometrial Biopsy
  or
  Endometrial Aspiration Cytology (EAC) } OPD Procedure ; NO Anesthesia ; NO Preparation
  ↳ preferred
  ↓
  using KARMAN'S CANULA / PIPELLE VABRA Aspirator.





- Gold Standard Procedure : D and C + HYSTEROSCOPY
  ↓
  Fractional curettage
  ↓
- Indications

1. Stenosed cervix
2. OPD Procedure : Benign Report but patient has persistent PMB
3. OPD Procedure : No Endometrial tissue obtained Only Blood and mucous
4. OPD Procedure : Report - HYPERPLASIA c̄ ATYPIA

- PAPs is also done for PMB

- Endometrial Sampling done in :

1. PMB
2. ≥ 45yrs c̄ AUB
3. If < 45yrs PCOS (Prolonged Amenorrhoea)
4. HNPCC : if K/c/o LYNCH Synd - TAH c̄ BSO
5. COWDEN Syndrome
6. GRANULOSA CELL Tumor

STAGING FOR ENDOMETRIAL CANCER [FIGO - Surgical]

- STAGE 1 -

A - If only endometrium involved or 50% of myometrium is involved

B - >50% Myometrium

- STAGE 4 -

A - Bladder / Rectum

B - Distant Spread Inguinal LN

- STAGE 2 - Cervical stroma (only if stroma involved)
- STAGE 3 -

A - Uterine serosa and/or adnexa

B - vagina and/or Parametrium

C - C_1 · ⊕ Pelvic LN
- C_2 · ⊕ Paraaortic LN

- BEST RADIOLOGICAL MODALITY - MRI
- Staging Laparotomy

① Omenectomy NOT DONE
↳ done in TYPE 2 HISTOLOGY

- SELECTIVE LYMPHADENECTOMY

↓

1. Gross Tumor >2cm
2. >50% Myometrium
3. Type 2 Histology (Non-Endometroid)
4. Cervical spread

② Cervical spread → TYPE 3C HYSTERECTOMY

NEW Type Ia
- Robotic / Laparoscopic Hysterectomy
- Sentinel LN Sampling done

↳ done in CA Vulva > CA Endometrium > CA Cervix

POST OP TREATMENT

1. LOW RISK - all the following criteria : ① Endometriod ② Only Endometrium ③ Grade 1 } NO Post OP Treatment Required

2. INTERMEDIATE RISK - Pelvic Radiotherapy in the form of EBRT

3. HIGH RISK - any of the any of the following ① Stage 3/4 ② Type 2 Histology } Rx. Chemo + Radio ↓ CISPLATIN + PACLITAXEL

- POOR PROGNOSTIC MARKER : TYPE 2/GRADE 3/ ↑ Stage [ER / PR ⊕ve - Good prognosis]

PROLAPSE

- descent of pelvic organs into vagina or even outside
- Supports of Uterus - ④ main
  1. Levator ani ⟶ Pubococcygeus and Iliococcygeus
  2. Uterosacral
  3. Cardinal
  4. Pubocervical

  } aid in maintaing the position

- Round ligament : Support, but not main
- Broad ligament : Not support
- Angle of Anteversion - 90° (Between Cervix and Vagina)
- Angle of Anteflexion - 120° (Between Body of Uterus and Cervix)
- Fundus of Uterus towards Bladder : ANTE FLEXED
- Ⓝ Position - ANTEVERTED and ANTEFLEXED - S/i 80% of women

↓ Ligament Responsible : Round ligament + Uterosacral ligament

DELANCEY'S LEVELS OF SUPPORT

LEVEL 1 : UTEROSACRAL + CARDINAL LIGAMENTS ENTEROCOELE

- if weak → uterocervical descent / Vault prolapse aka APICAL PROLAPSE

1st Degree : descent from vaginal position but are still above vagina

2nd Degree : reach the vaginal introitus

3rd Degree : Outside the vagina

4th Degree : PROCEDENTIA - entire uterus lies outside

LEVEL - 2 : Paravaginal tissue and its attachment to fascia of Levator ani & White Line

- if becomes weak → protrusion of bladder [CYSTOCOELE] : Bulge in upper 2/3rd of Anterior Vaginal wall
- (MC) Type of PELVIC ORGAN PROLAPSE

LEVEL 3 : Perineal Body + Muscles attached to it

- if becomes weak → RECTOCOELE → lower 2/3rd of Posterior Vaginal Wall
- Bowel loops protrude through POD - upper 1/3rd of Posterior Vaginal Wall

VAULT : Apex of vagina in Posterior Hysterectomy patients

RISK FACTOR For PROLAPSE

- ↑ Age and Parity (↓ ESTROGEN)
- also seen in Young (Reproductive Age group)
  - ① Connective Tissue disorder
  - ② Spinal cord Injury
  - ③ c/c ↑ Intra Abdominal pressure
  - ④ Congenital Elongation of Cervix

- SYMPTOMS : 1. Dragging sensation - Something coming out from vagina

  2. Urinary Symptoms - Stress incontinence ; difficulty in passing urine

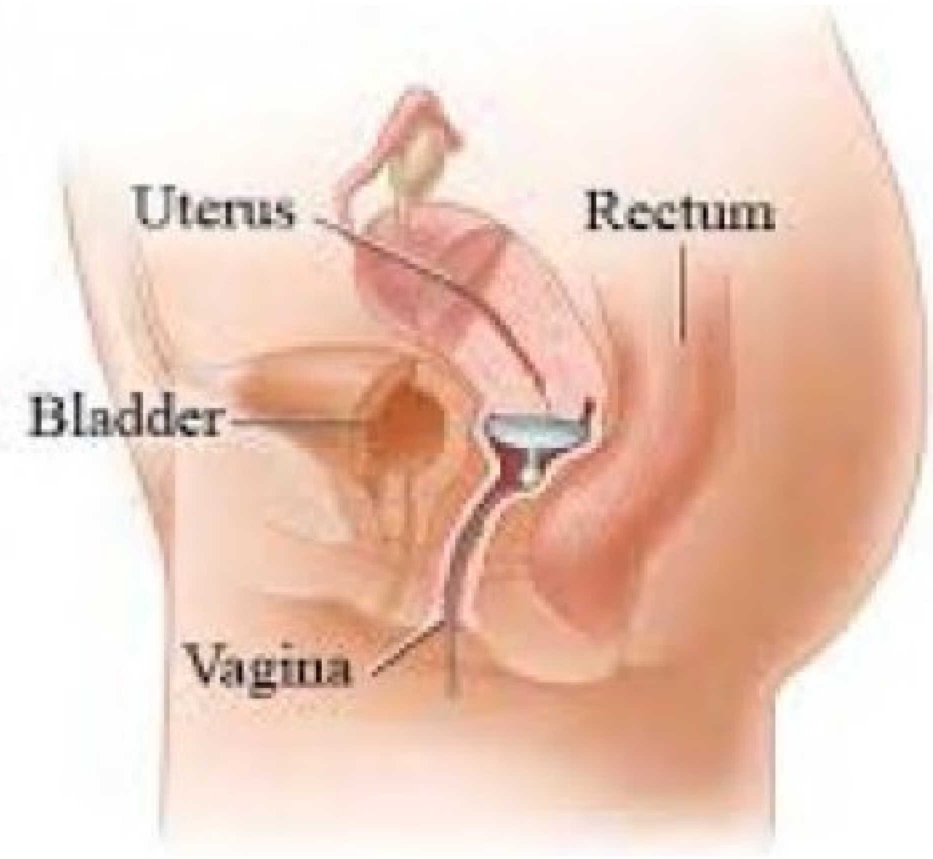
3. Bowel Related Symptoms   4. Discharge / Bleeding P/v h/o

- DECUBITUS ULCER (s/i 3rd Degree) - venous congestion

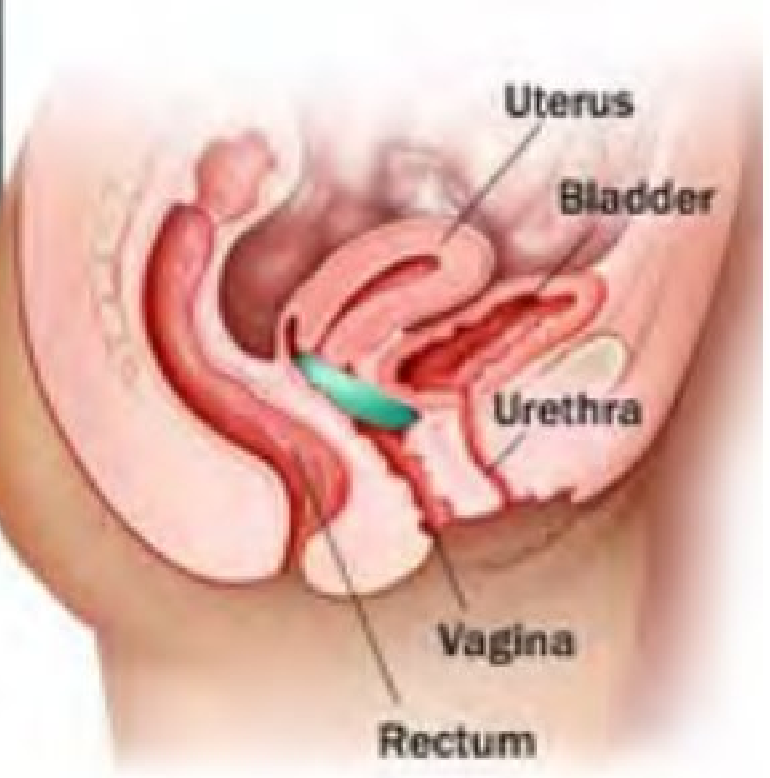
NON-OPERATIVE MANAGEMENT

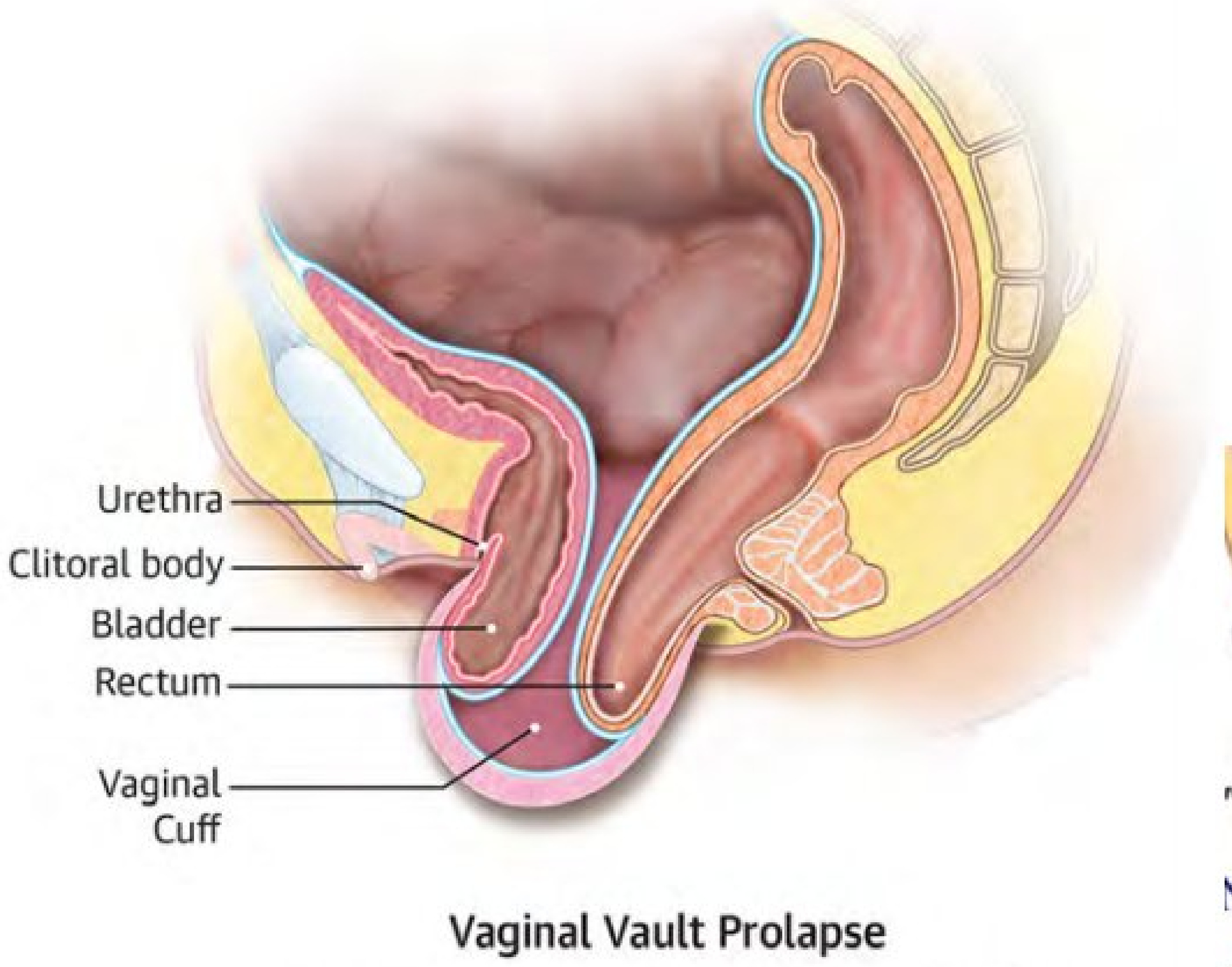
1. KIEGEL'S EXCERCISE :
strengthen muscles of pelvis

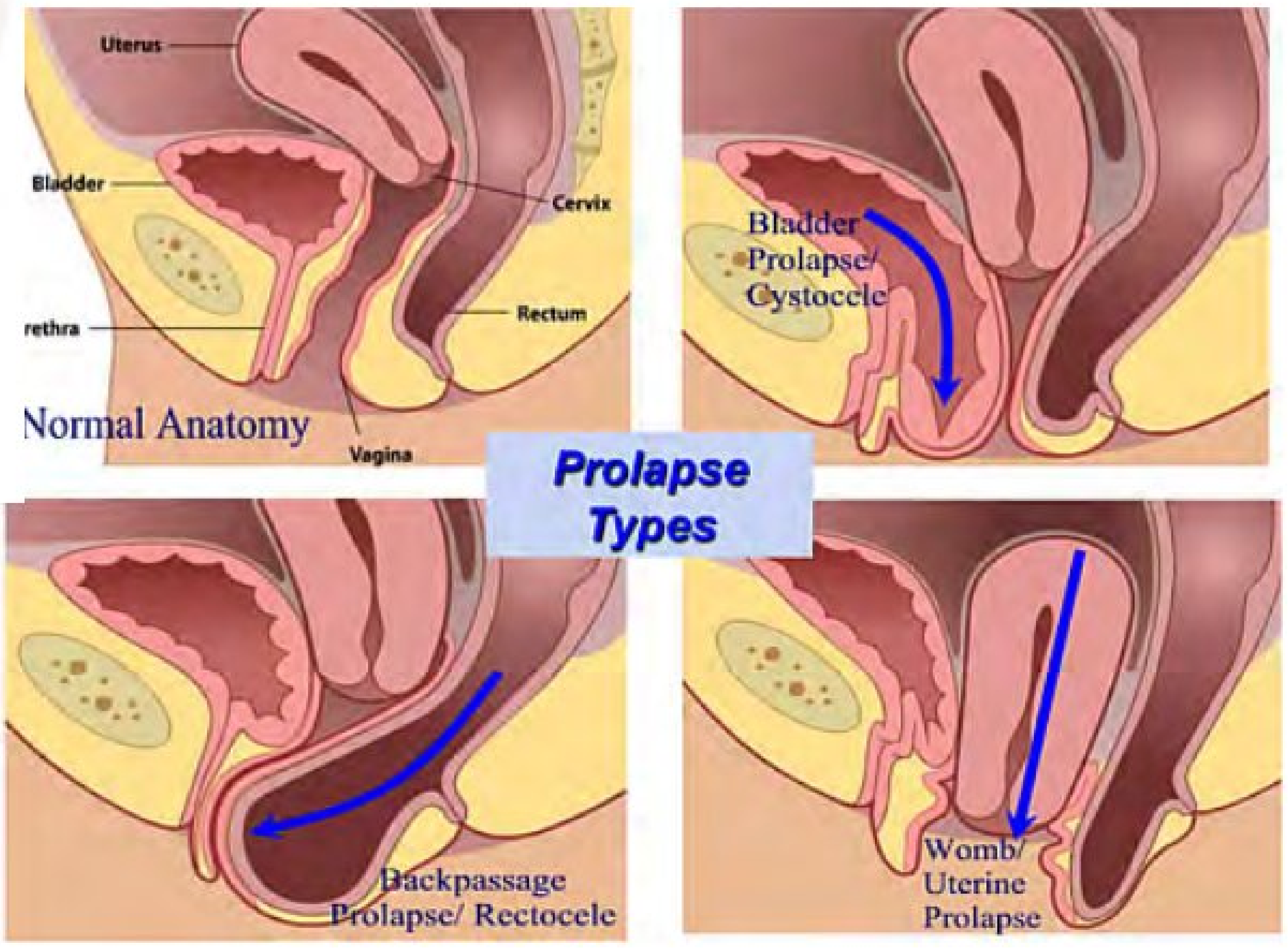
2. PESSARY -
- DOUGHNUT - use for cystocoele
- GELL HORN - used in Utero Cervix descent



used in patients :
i) Refuses Surgery
ii) Unfit for Surgery
iii) Pregnancy c̄ Prolapse / in Puerperium
↳ Remove it around 16-18 wks







SURGICAL TREATMENT: depends on Age group

Q. 60yr old P_3L_3 3rd degree prolapse + cystocoele + Rectocoele

- for Utero cervical descent - Vaginal Hysterectomy

↳ Ligation : 1st - ① Uterosacral ligament
② Cardinal ligament
③ Uterine artery
④ Utero-ovarian pedicle

- In TAH: Ligation → ④ → ③ → ② → ①

- TAH → Utero cervical
- SUBTOTAL → Supracervical
- TAH + BSO → PAN HYSTERECTOMY.

URINARY TRACT INJURIES

- Risk c̄ HYSTERECTOMY : Modified Radical or Radical > Lap > Abdominal > Vaginal

  Modified Radical or Radical — done in Malignancy

  Lap > Abdominal > Vaginal — done in BENIGN

- for cystocoele : Anterior colporrhaphy
- for Rectocoele : Posterior colpoperineorrhaphy (suture from levator ani)

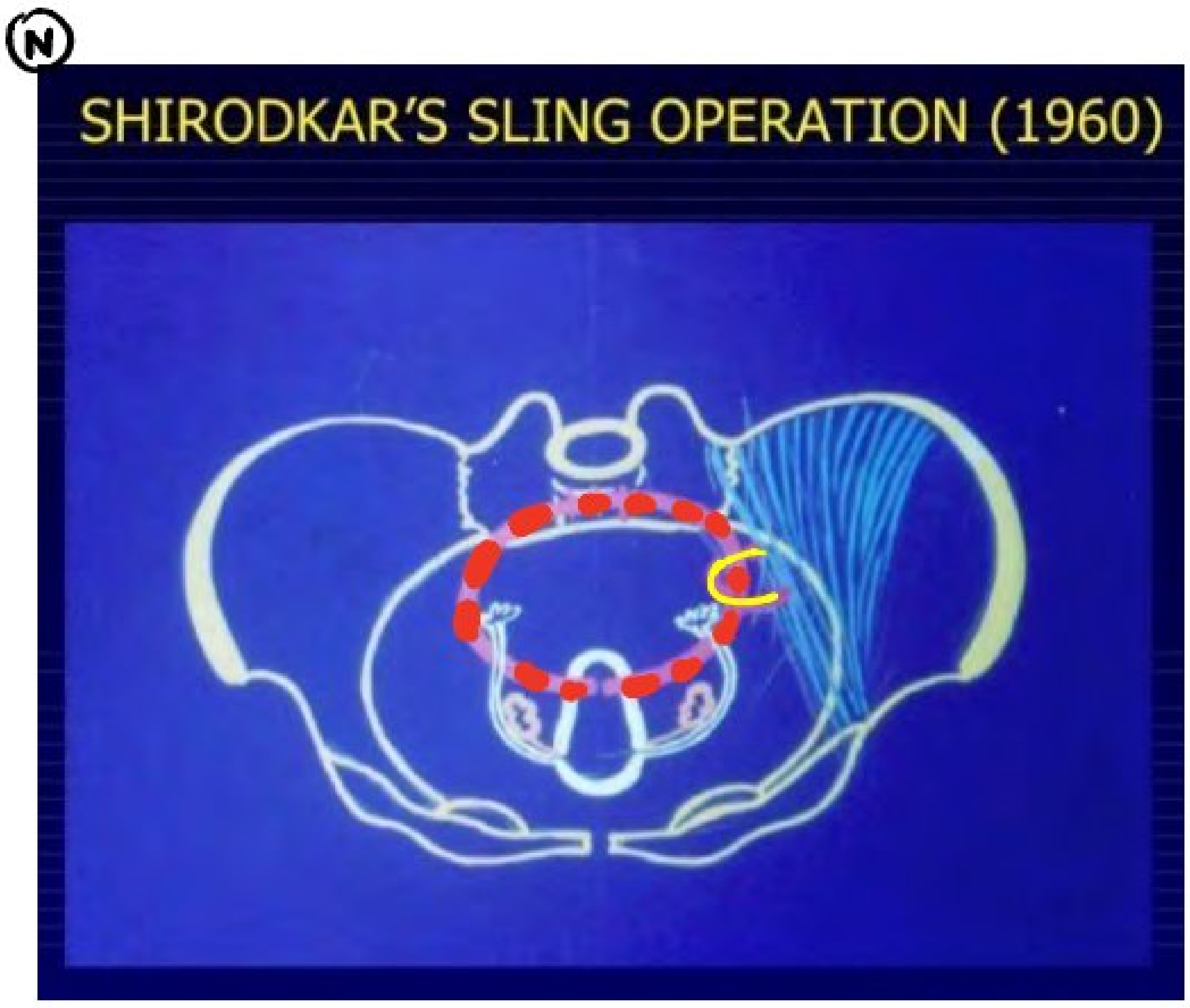
- VH + Anterior colporrhaphy + Posterior colporrhaphy } VH + PFR (Pelvic Floor Repair) } WARD MAYO Surgery

- For ENTEROCOELE : VH + PFR +
  1) McCalls Culdoplasty – done prophylactically in
     a) Enterocoele
     b) Vault prolapse
     ↳ by uterosacral suspension
  2) MOSCHOWITZ Repair
  3) HALBAIN'S

- In REPRODUCTIVE AGE Group
  - TOC : SLING SURGERIES

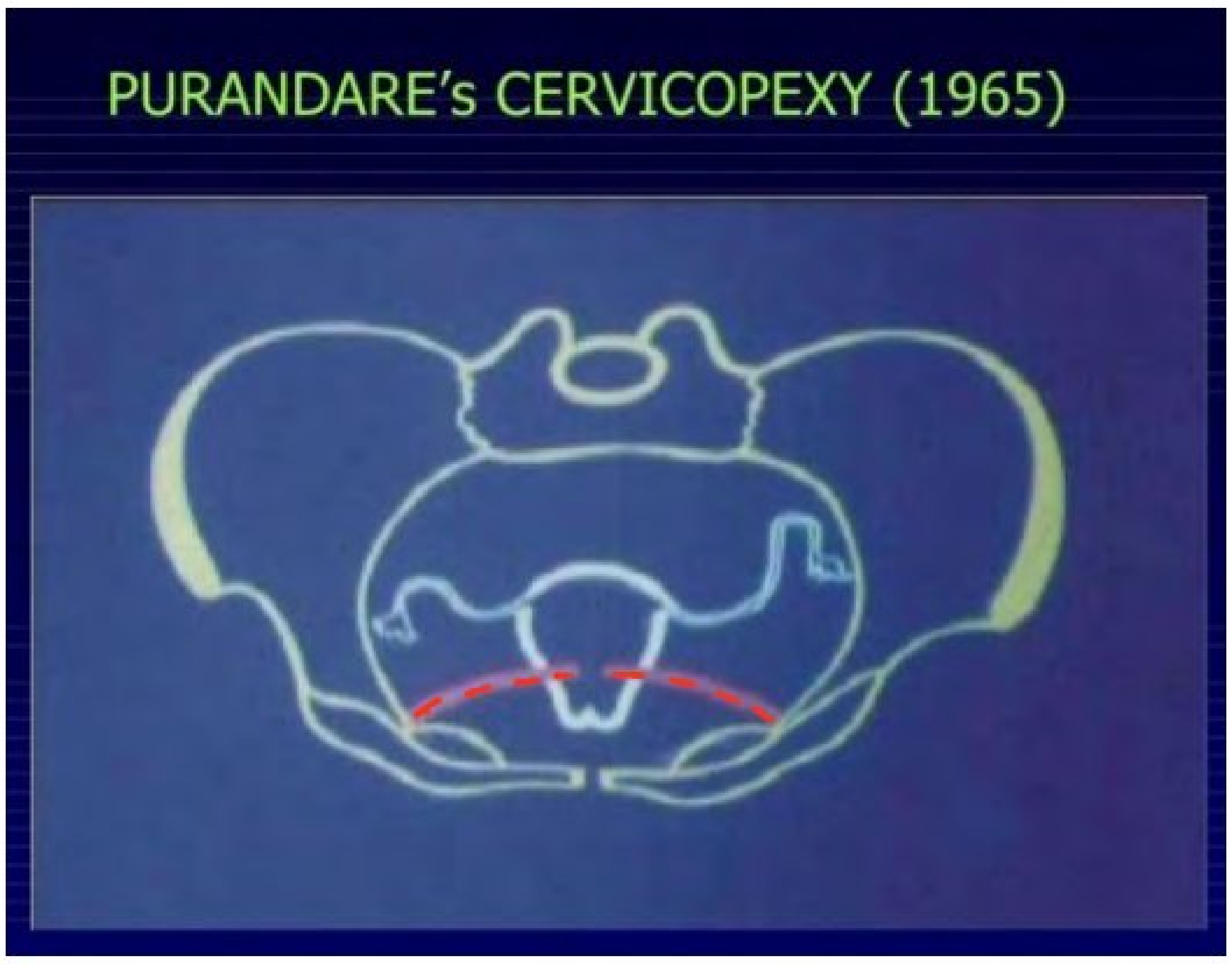
ANTERIOR SLING	POSTERIOR SLING
Anterior Isthmus	Posterior Isthmus
Tensor Fascia lata	Merselene Tape
Anterior Abdominal wall (External Oblique Aponeurosis)	ASIS
PURANDRE'S SLING	KHANNA'S SLING

1. SHIRODKAR ABDOMINAL SLING – UCL should be Ⓝ

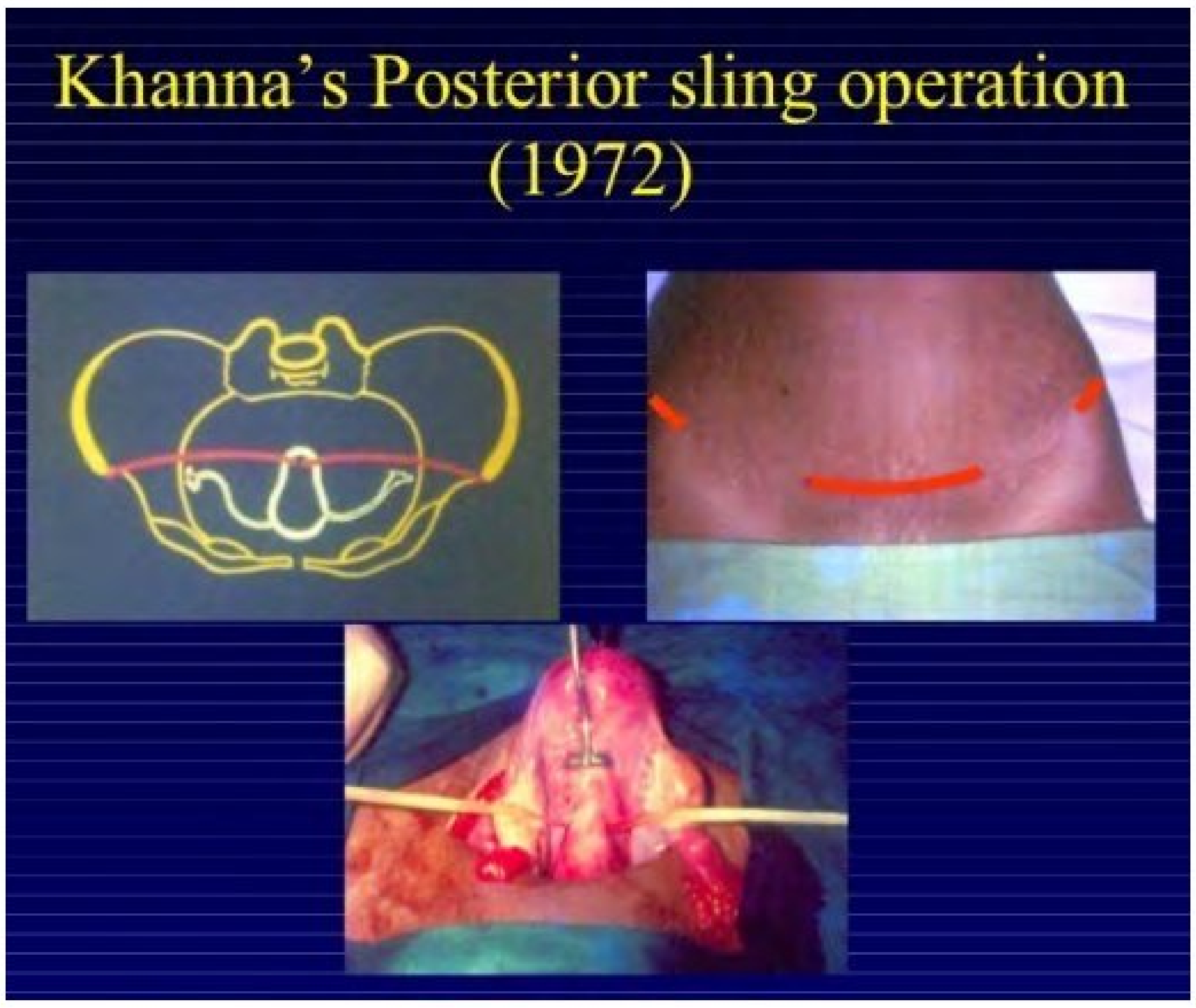

SHIRODKAR'S SLING OPERATION (1960)

- Abdominal Surgery
- Merselene tape used
- Post. Sling Surgery – TOC for Reproductive women

Ⓛ Side	Ⓡ side
Through PSOAS Hook	Post isthmus
Sacrum	Sacrum



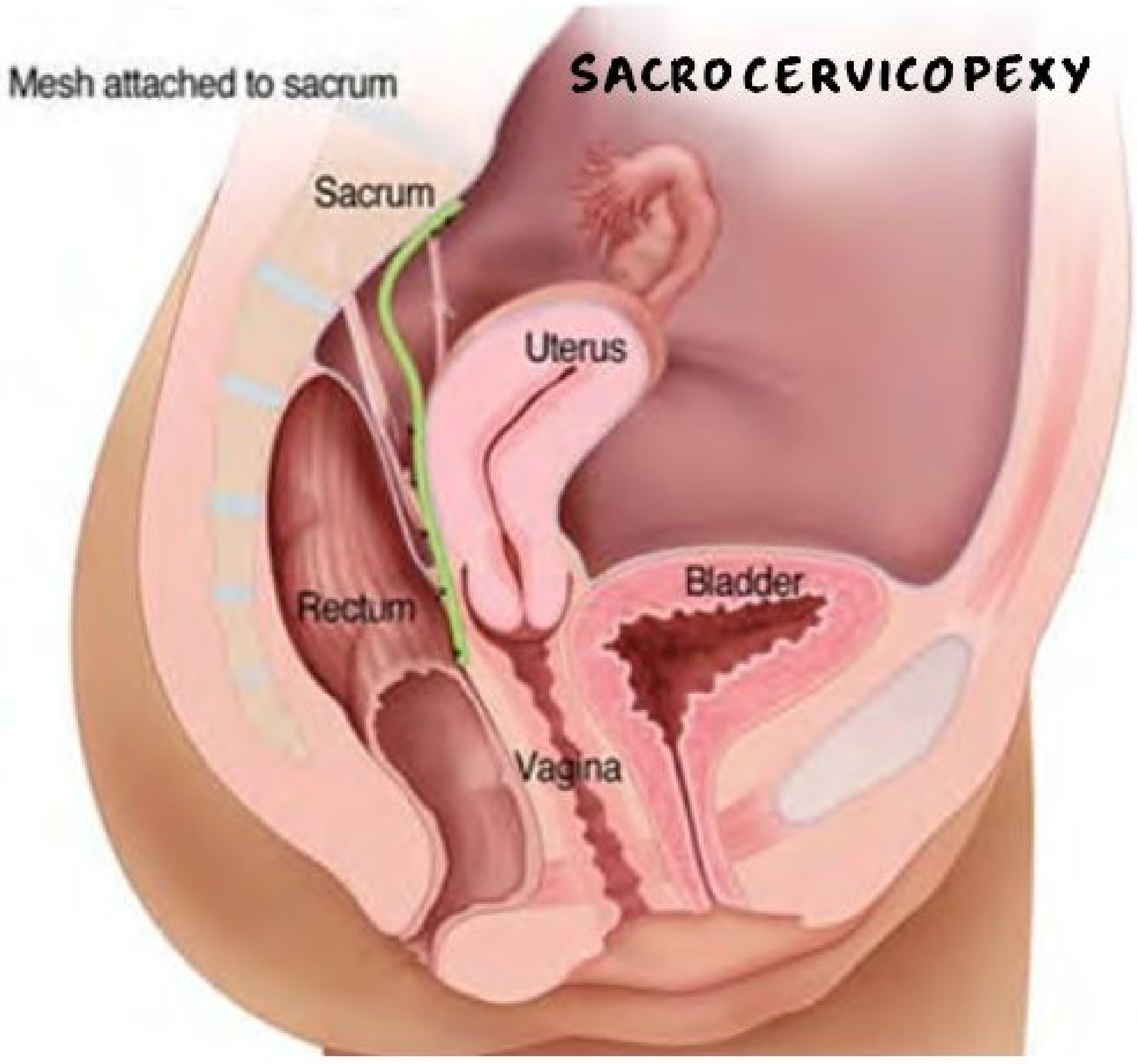
PURANDARE's CERVICOPEXY (1965)



Khanna's Posterior sling operation (1972)

2. SACROCERVICOPEXY

- mesh used ; kept b/w sacrum (post) and Uterus, cervix (Anteriorly)
- Mesh undergoes FIBROSIS and permanently attached to sacrum



SACROCERVICOPEXY

3. MANCHESTER REPAIR aka FOTHERGILL'S REPAIR

- NO Sling used
- cervical amputation ; reattach Cardinal ligament anteriorly

  ↓

  can cause 1) Stenosis of Cervix 2) Incompetence
- done if family complete ; Congenital Cervix elongation

4. SHIRODKAR'S MODIFICATION OF MANCHESTER

- cervical amputation – not done
- done if family is INCOMPLETE
- Uterosacral length : Ⓝ 6-8cm

Q. 3rd Degree Prolapse ; but she have co-morbidities fit for Short Surgeries. What next?

Ans : **LE-FORT COLPOCLEISIS**

- Remove mucosa from Anterior wall, Posterior wall
- Before doing this R/o CA Cervix and CA Endometrium
- Pt can't be sexually active after procedure

VAULT PROLAPSE : s/i POST-HYSTERECTOMY Patients

- Surgeries done : I. VAGINAL - (MC) done
  - 1. SACROSPINOUS FIXATION → Sutures b/w vaginal vault and Sacrospinous ligament
  - 2. UTEROSACRAL SUSPENSION

II. ABDOMINAL - 1. SACROCOLPOPEXY - BEST OUTCOME

2. UTEROSACRAL SUSPENSION

COLPOSUSPENSION : • Surgery done for STRESS URINARY INCONTINENCE → TOC : TVT / TOT

aka BURCH COLPOSUSPENSION → Abdominal Surgery support proximal part of Urethra
[Gold Std for SUI]

- TVT - Trans Vaginal Tape
- TOT - Trans Obturator Tape

} Sling Surgery for SUI
Middle Urethra supported } aka MID URETHRAL SLING SURGERY

TVT → Retro pubic Sx
(RETZIUS Space)

- Both Surgeries are Daycare Surgery ; Vaginal Surgery
- as good as Burch Colposuspension
↓
- Support Only penile urethra - COOPER's LIGAMENT
- not an eg. for Sling Surgery

POPQ (Pelvic Organ Prolapse Quantification) : New classification System for Prolapse.

FIBROID

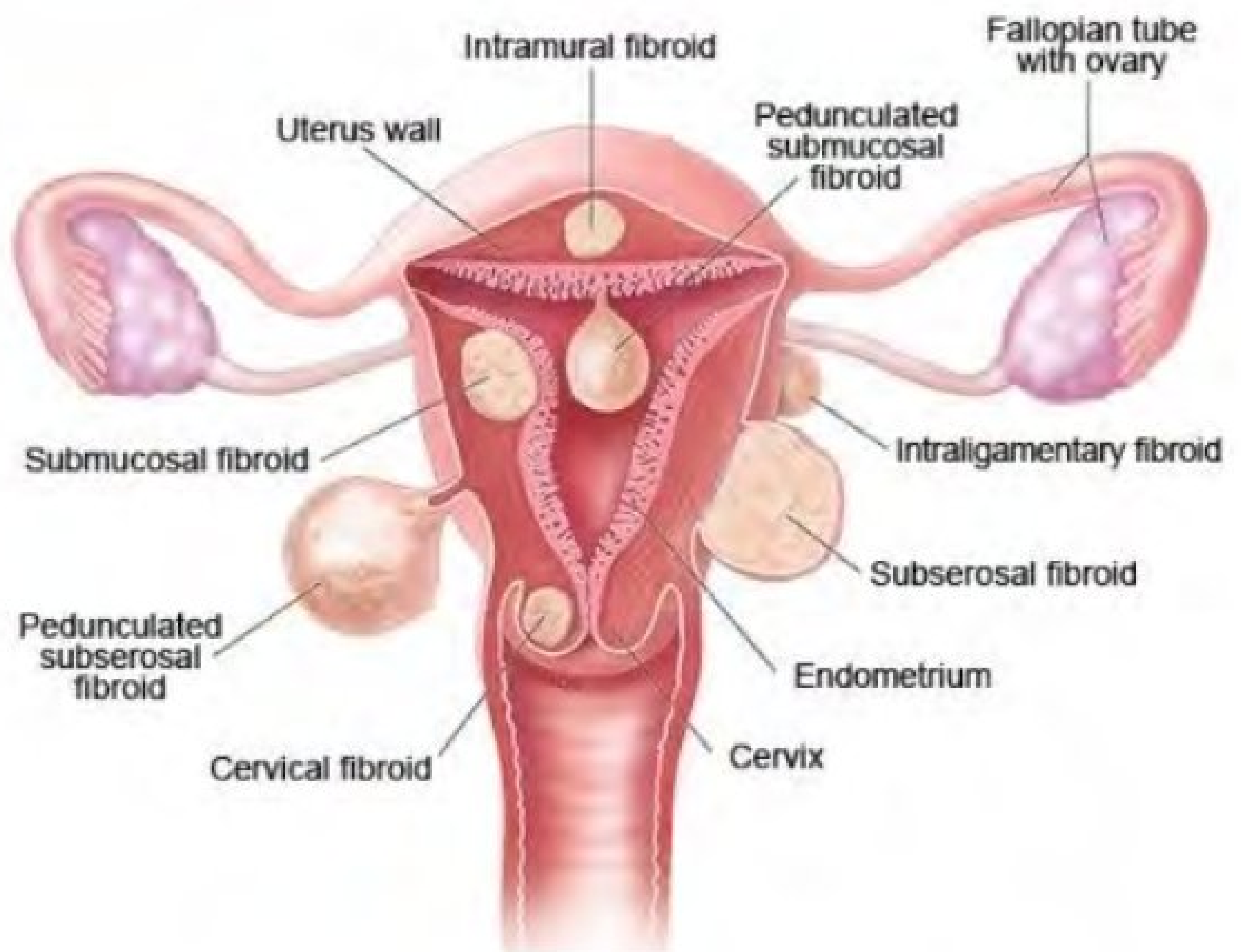
- Smooth muscle tumor of Uterus
- (MC) Benign Pelvic Tumor in Females
- ESTROGEN dependent.
- Reproductive Age (>35yrs)
- (MC) Presentation : ASYMPTOMATIC
- (MC) Symptom : I. INCREASED BLEEDING / MENORRHAGIA (x irregular cycles)
  (> 200ml ; s/i 40% pt)
- (MC) Fibroid - INTRAMURAL (75%)
- (LC) Fibroid - SUBMUCOSAL (10%) - MOST SYMPTOMATIC

FIGO CLASSIFICATION : 0, 1, 2 → SUBMUCOSAL
3, 4 → INTRAMURAL
5, 6, 7 → SUBSEROSAL → become PARASITIC LEIOMYOMAS
8 → EXTRA UTERINE FIBROID

0 - Completely Intracavitary } can be removed
1 - >50% Intracavitary

HYSTEROSCOPICALLY

2 - <50% Intracavitary



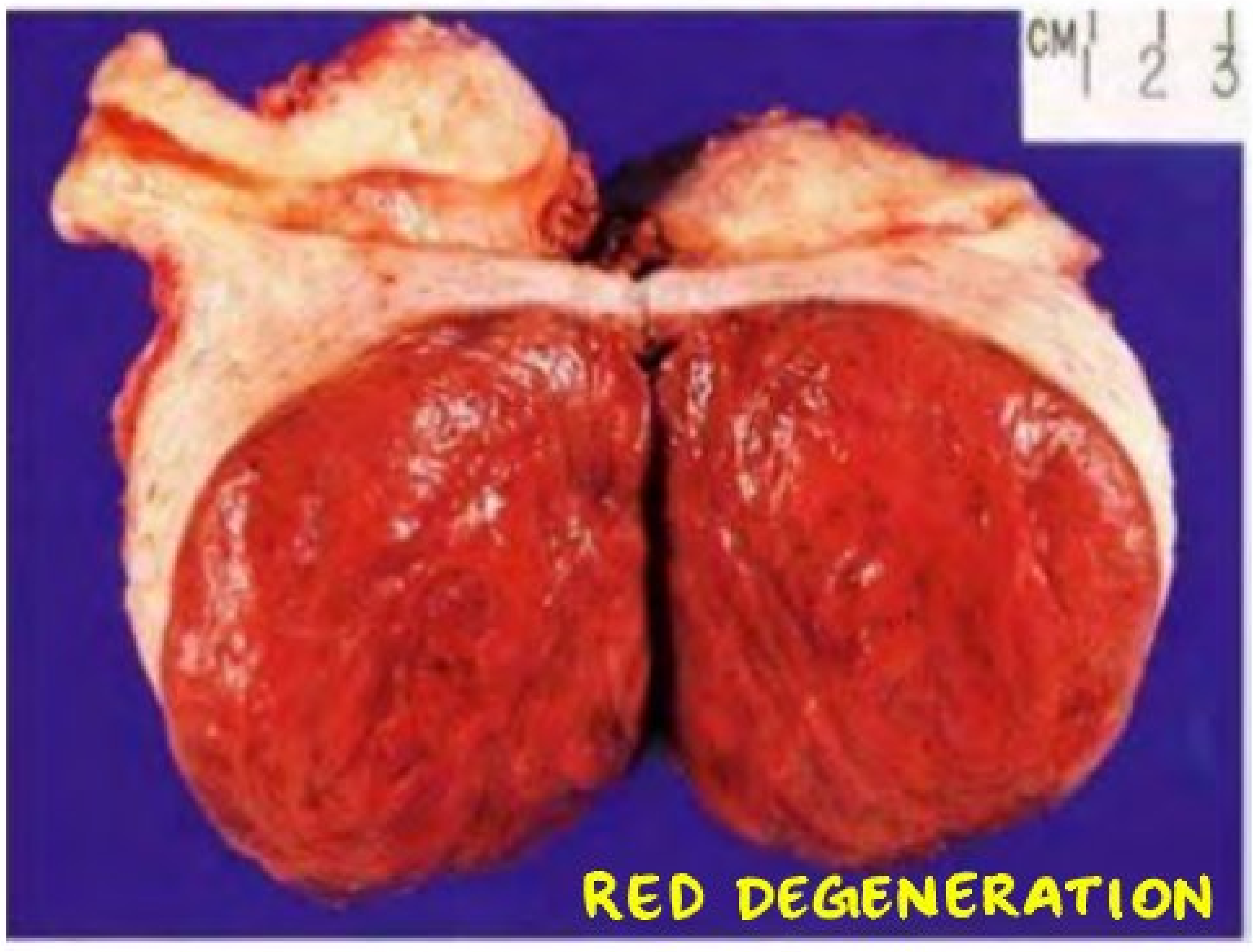
II. PAIN → if fibroids undergo DEGENERATION, TORSION ↗ pedunculated Subserosal fibroid

- Degeneration begins usually at centre ; Calcification s/i Periphery
- (MC) degeneration - HYALINE DEGENERATION (60%) - occurs in Subserous
- (LC) degeneration - SARCOMATOUS Degeneration - Rare Malignant change
- <0.5 % - Post menopausal → Fibroid grows ↑ Painful
- Calcific degeneration -

(MC) s/w SUBSEROSAL → D/D Bladder Stone → Symptomatic
↳ Incidental finding asymptomatic - ECCENTRIC "WOMB STONE"

RED DEGENERATION : pregnancy ; painful and Red

- s/i 2nd TM
- A/c Abdomen - pain, Nausea, Vomiting, ± fever
- Conservative Rx: NPO, i.v fluids, Analgesics
- In Pregnancy : most fibroid donot grow in size



III. PRESSURE - (MC) ≤ Sensorineural Bladder and Bowel

- Anterior Wall of Uterus : ↑ Frequency of MICTURITION
- Posterior Wall of Uterus : Retention of urine

IV. ABORTIONS   V. INFERTILITY

IOC : USG → Pseudocapsule d/t this well demarcated "HYPOECHOIC"

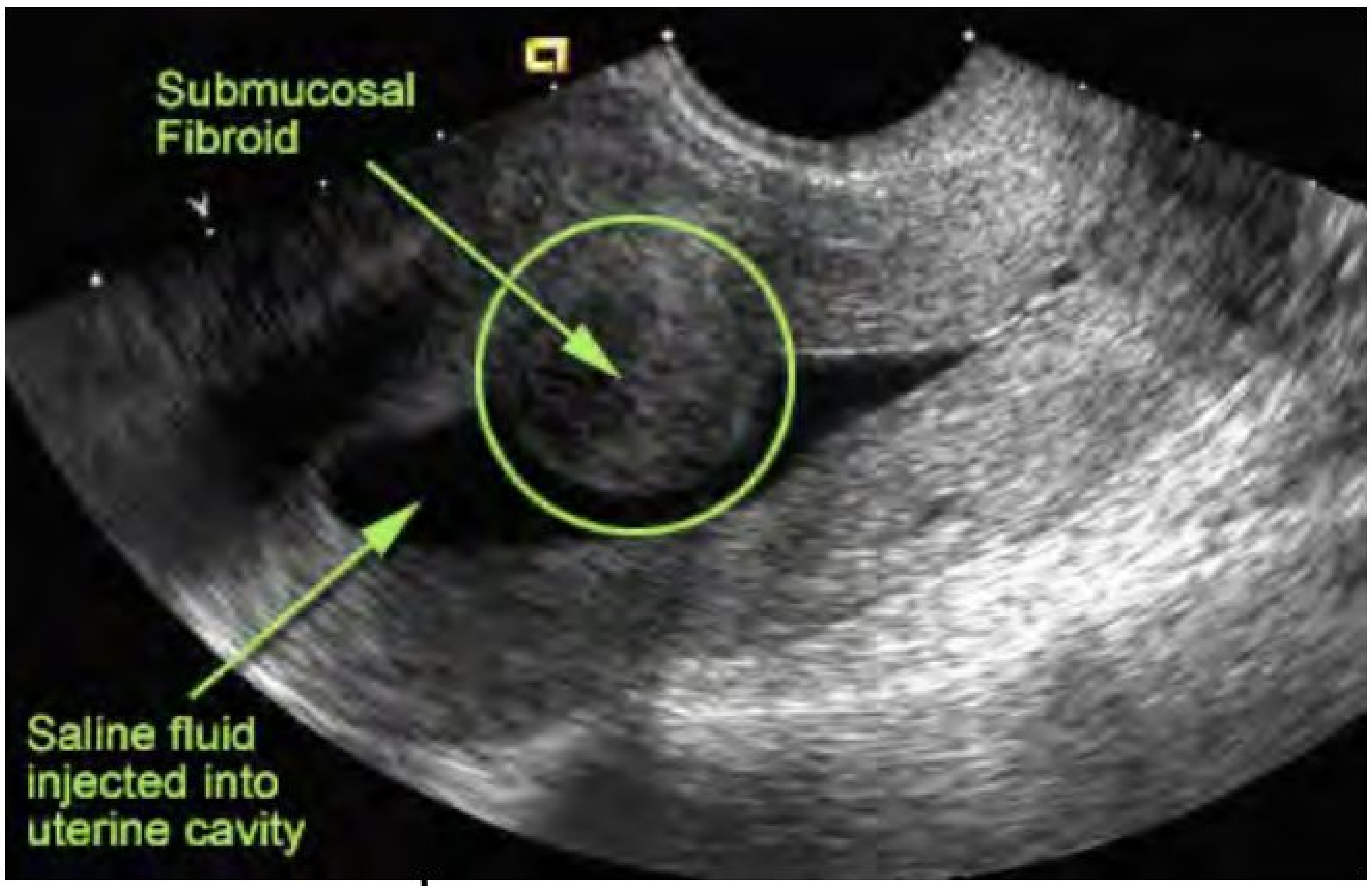
In Pregnancy can cause :
1. ABORTION
2. RED DEGENERATION
3. PRE-TERM LABOUR
4. ABRUPTION
5. MALPRESENTATION
6. PPH

- small Submucosal fibroids ↓

MISSED On USG

So, BEST Invg: HYSTEROSCOPY.

SIS : Saline Infusion Sonography
(non- invasive)



FIBROID	POLYP
• Broad Base • From Myometrium	• Thin Base
PERIPHERY ← Do Doppler	→ CENTER
	Rx. HYSTEROSCOPIC POLYPECTOMY

ADENOMYOSIS

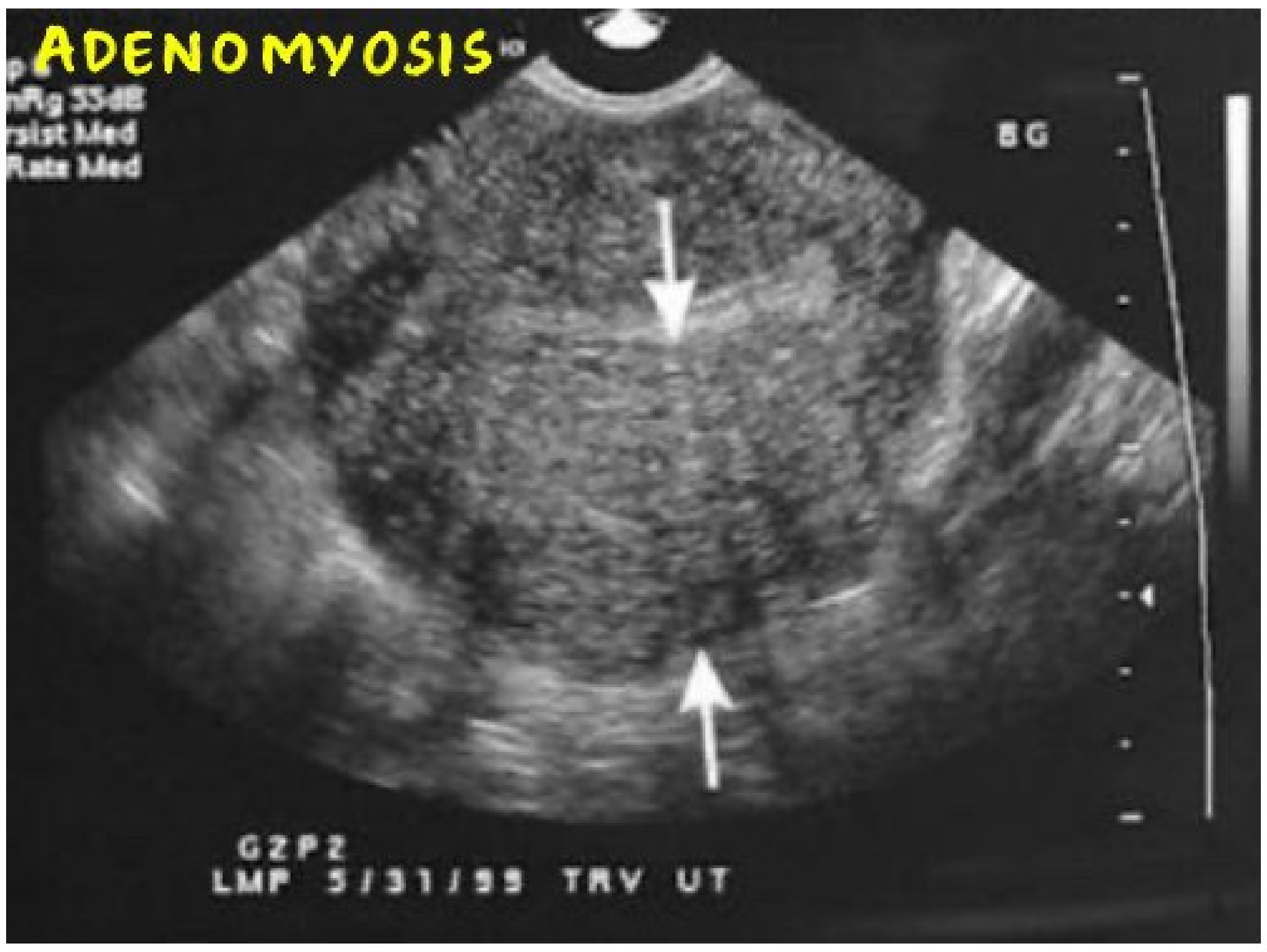
- Endometrial gland and stroma c̄ in MYOMETRIUM

C/F • Menorrhagia a/w DYSMENORRHOEA (40-45yrs)

- Symmetrical enlarged uterus, globular; Bleeding points (d/t withdrawal)
- tender uterus
- growth 10-12wks size

Invg: 1st USG but IOC - MRI : Endo-myometrial junction zone THICKNESS - ≥12mm s/o Adenomyosis
↓

- Heterogenous myometrium "SALT and PEPPER" App.
- Confirm Diagnosis c̄ HPE (Post. Hysterectomy)



RxOC : HYSTERECTOMY

TREATMENT OF FIBROIDS

- Only for Symptomatic fibroids : Menorrhagia
- DOC : OCP, MIRENA, TRANEXA } ↓ Blood loss but doesnot reduce Size
- Drugs which ↓ Size, ↓ Blood loss.

① GnRH Agonist : should be given continuously. If stopped will Regrow. Can't be given for long duration

- given Pre-OP because it reduce intra OP blood loss
- Dis Adv : fibroid becomes difficult removal ; planes of dissection lost.

2. **GnRH Agonist :** can be used

3. **MIFEPRISTONE :** Anti PG's → ABORTIFACIENT
4. **ULIPRISTAL :** SPRM (Selective Progesterone Receptor Modulator) emergency Contraceptive

} Not used in women in Reproductive Age grp.

5. **AROMATASE INHIBITORS**

Medical Management
↓
No Response → and NOT WILLING for Surgery → MINIMALLY INVASIVE METHODS (Uterine Artery Embolization)
↓
do Surgical Management

NON - INVASIVE :

MRgHIFU (Magnetic Resonance Guided High Intensity Focussed USG)

- OPD procedure
- Relief of symptoms Immediate
- Fibroid doesnot REGROW

C/I ① > 10cm ② > 5 in Number ③ Calcified ④ Hysteroscopically removed.

SURGICAL MANAGENT

1. CONSERVATIVE - family is desired and to preserve organ (Myomectomy > Myolysis)

MYOMECTOMY : 0, 1 - Submucosal - Hysteroscopic myomectomy

Type 2 Hysterectomy → Intramural } Lap >> Abd
→ Subserosal

MYOLYSIS : fertility NOT IMPROVED

2. RADICAL - HYSTERECTOMY when family is complete

VAGINITIS / VAGINAL DISCHARGE

BACTERIAL	CANDIDIASIS	TRICHOMONAS
• Gardenella vaginalis (Mycoplasma mobilincus Ureaplasma Peptostreptococcus) • pH > 4.5 • foul smelling discharge	C. albicans pH < 4.5 • pruritus (mainly) • Scanty Discharge ; because it sticks to vaginal wall	T. vaginalis pH > 4.5 • foul smelling discharge

BACTERIAL	CANDIDIASIS	TRICHOMONAS
• Grey / off white discharge • Pruritis - No • Urinary symptom - No • Dyspareunia - No	• Curdy white Yes Splash dysuria Yes	• Green / Yellow +/- ↑ frequency / dysuria Yes
IOC for VAGINAL DISCHARGE : SALINE MICROSCOPY		
• Clue cells - Epithelial cells loaded c̲ Bacteria	• Pseudohyphae	• flagellated organism seen
BEST INVESTIGATION		
GRAM STAIN AMSEL'S CRITERIA - 3/4 1. pH < 4.5 2. Grey off white foul smelling discharge 3. Clue cells (20% Epithelial) 4. WHIFF Test - POSITIVE • On 10% KOH - Fishy odour • Not a STD, so NO PARTNER treatment requirement TREATMENT : Metronidazole Clindamycin	SDA Culture • Not a STD, Partner is treated if he is symptomatic TREATMENT: Single oral dose 150mg Flucconazole	STUART's Media Culture • STD, Partner Treatment must • TREATMENT : Metronidazole

FISTULA

- MC Type - UROGENITAL FISTULA (VVF)
- MC cause in developing countries - OBSTRUCTED LABOUR
  - developed countries - GYNAECOLOGICAL SURGERY
- Late complication - (7-10 days Post Surgery) = 2nd wk
- Diagnosis : 3 SWAB Test.
- Retrogradely fill Bladder c̲ dye

0 > upper 2
0
0 > lower 2

- upper 2 swabs are 'wet' and 'stained'
- if along c̲ lower swab is 'wet' and 'stained' [URETHRO VAGINAL FISTULA]
- if only upper swab 'wet' but not 'stained' [URETERO VAGINAL FISTULA]

- If h/o continuous leakage of urine - No Ⓝ voiding - VVF
- If h/o continuous leakage and also Ⓝ voiding - Uretero vaginal fistula

BEST Investigation VVF : CYSTOSCOPY - site, size, number

- Surgery for VVF - LATZKO Surgery.

Oogonia $\xrightarrow{\text{MITOSIS}}$ 1° oocyte (Diploid) $\xrightarrow[\text{(MI)}]{\text{MEIOSIS I}}$ get arrested in DICTYOTENE b/w Prophase and Metaphase → M I completed at ovulation

M I completed at ovulation → 2° oocyte [Haploid] ovum ; 1st Polar Body

2° oocyte $\xrightarrow{\text{MEIOSIS II}}$ Arrested in Metaphase Completed after Fertilization → 2nd Polar Body ; Female Pronucleus

CAPACITATION

- Begins in CERVIX, main site : FT, Duration : 7hrs, Motility - Epididymis
- Special motility : HYPERMOTILITY - altered memb permeability ; lot of influx to Ca^{2+}
  - gets the ability to undergo Acrosomal Reaction
  - gets the ability to bind to ZONA PELLUCIDA.

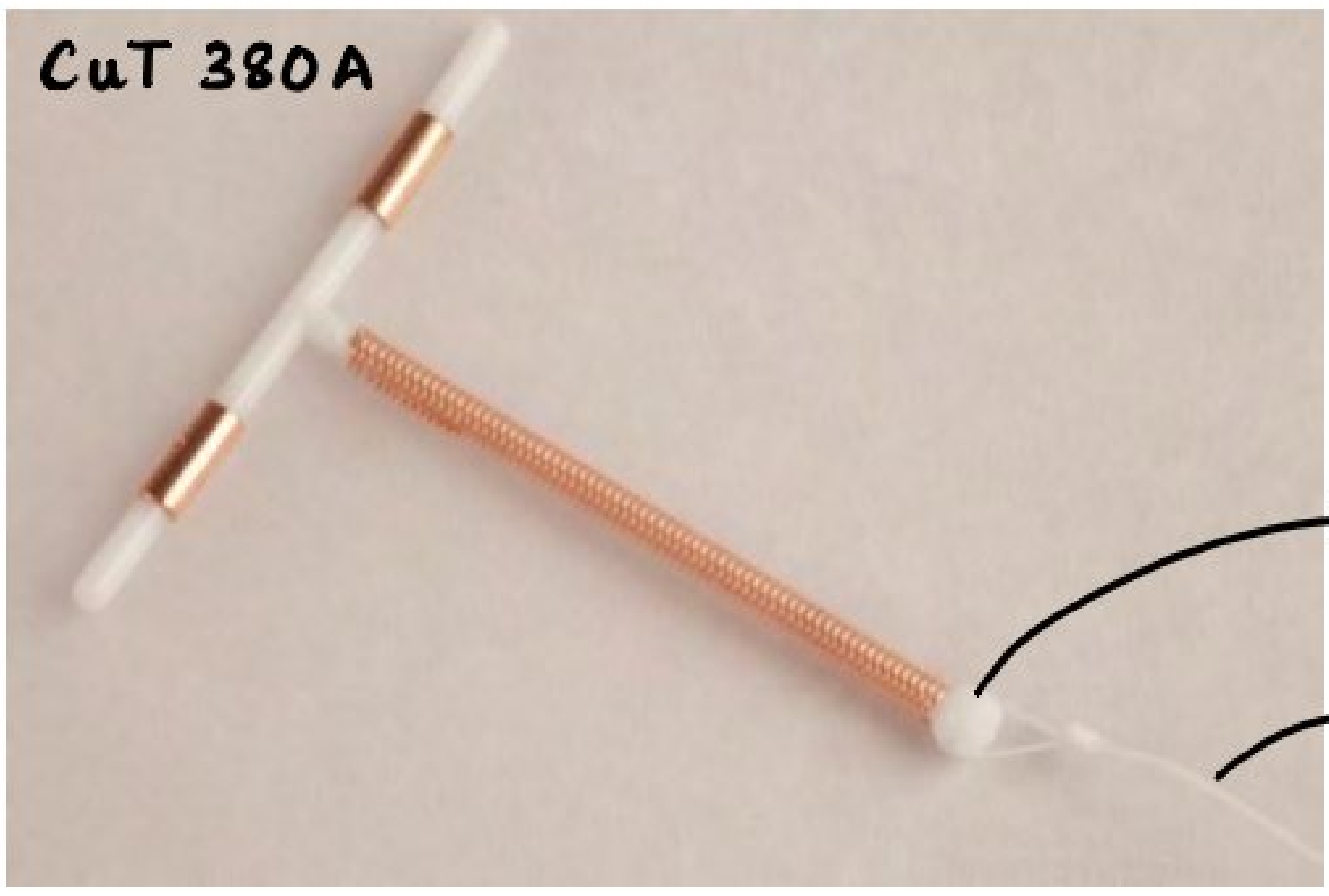
IUD - Intra Uterine Device

- commonly used / Reversible Rapidly
- Safe / Long acting
- User independent

1. CuT 380A
2. MIRENA
3. MULTI LOAD 375

1. CuT 380A [aka PARAGARD]

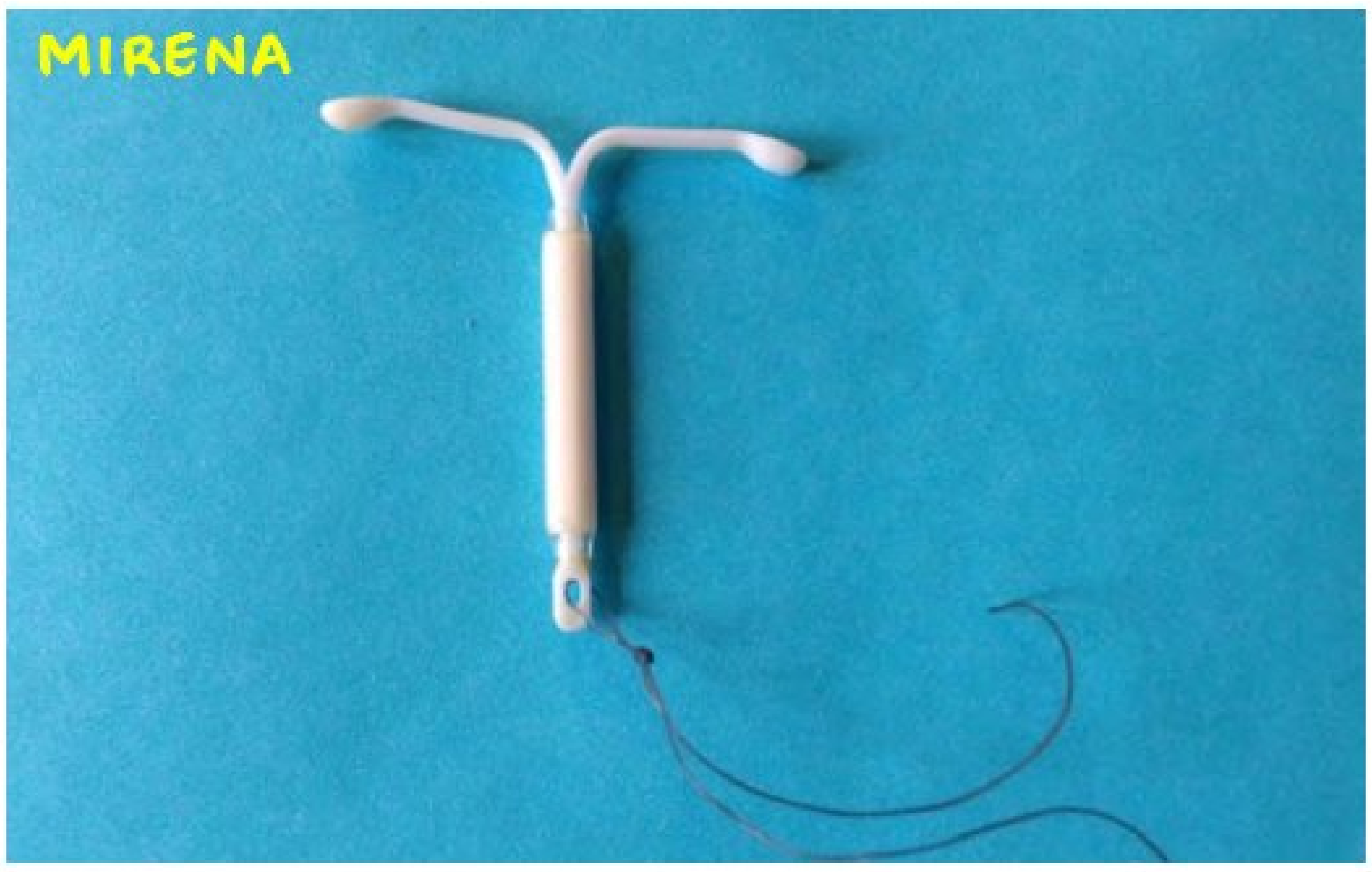
- frame 'T' Polyethylene
- Surface area : 380
- 'A' - Arms
- 2nd Gen IUD
- coated c̄ $BaSO_4$



3mm ball prevents cervical perforation

Monofilament threads

2. MIRENA



- poly dimethyl siloxane - around stem
- LNG - 52mg • Hormone Releasing
- drug delivery system only on vertical limb
- coated c̄ $BaSO_4$ - Radio opaque
- 3rd Gen. IUD - Efficacy comparable to sterilization.

	CuT 380A	MIRENA
	• Paragard • 380 Surface Area	• LNG 20 • 52mg
Release Rate	50mcg/day	20mcg/day
Life span	10yrs	5yrs
Drug Released in years	12yrs	7yrs
Free of cost	Yes	⊗
Emergency Contraceptive	Yes, most effective 120hrs or 5day	⊗
Bleeding	↑ menstrual blood flow by 50%	↓ • irregular bleeding • Amenorrhoea
Failure Rate (Perfect use)	0.6	0.1
Typical use	0.8	0.2

- In general : (MC) S/E - Bleeding (less c̄ 3rd Gen)
- (MC) cause of Removal : PAIN

MOA of IUD : Inhibits fertilization > Inhibition of Implantation (doesnot act by inhibition of ovulation)

POST-PARTUM IUD : inserted c̄in 48hrs of delivery; if not then by end of 6wks.

POST-PLACENTAL IUD : inserted c̄in 10min of Placental expulsion

- if threads are missing : IOC → USG → If not then do X-Ray Pelvis

IF WOMAN BECOMES PREGNANT WITH CuT IN-SITU ⟶ If threads are visible

↓

- Remove the CuT and she can continue pregnancy
- If left in-situ there is ↑sed Risk of
  ① Abortions ② Pre-Term Labour ③ Infection

if CuT is NOT VISIBLE : Leave it insitu

Could be ECTOPIC : do USG

ABSOLUTE CONTRAINDICATIONS

1. Undiagnosed Bleeding
2. Suspected Pregnancy
3. WILSON's Ds (absolute for CuT)
4. Severely distorted uterine cavity (Anomalies)
5. Ascitic pelvic infection; CA Cervix, CA Uterus, Adnexa and other pelvic tumors
6. Breast cancer (Absolute for MIRENA)
7. Severe Dysmenorrhoea / Menorrhagia
8. c̄ in 6wks of CS

- ↑ Risk of Infection c̄ IUD is only in 1st 20days post-insertion

CuT > Mirena

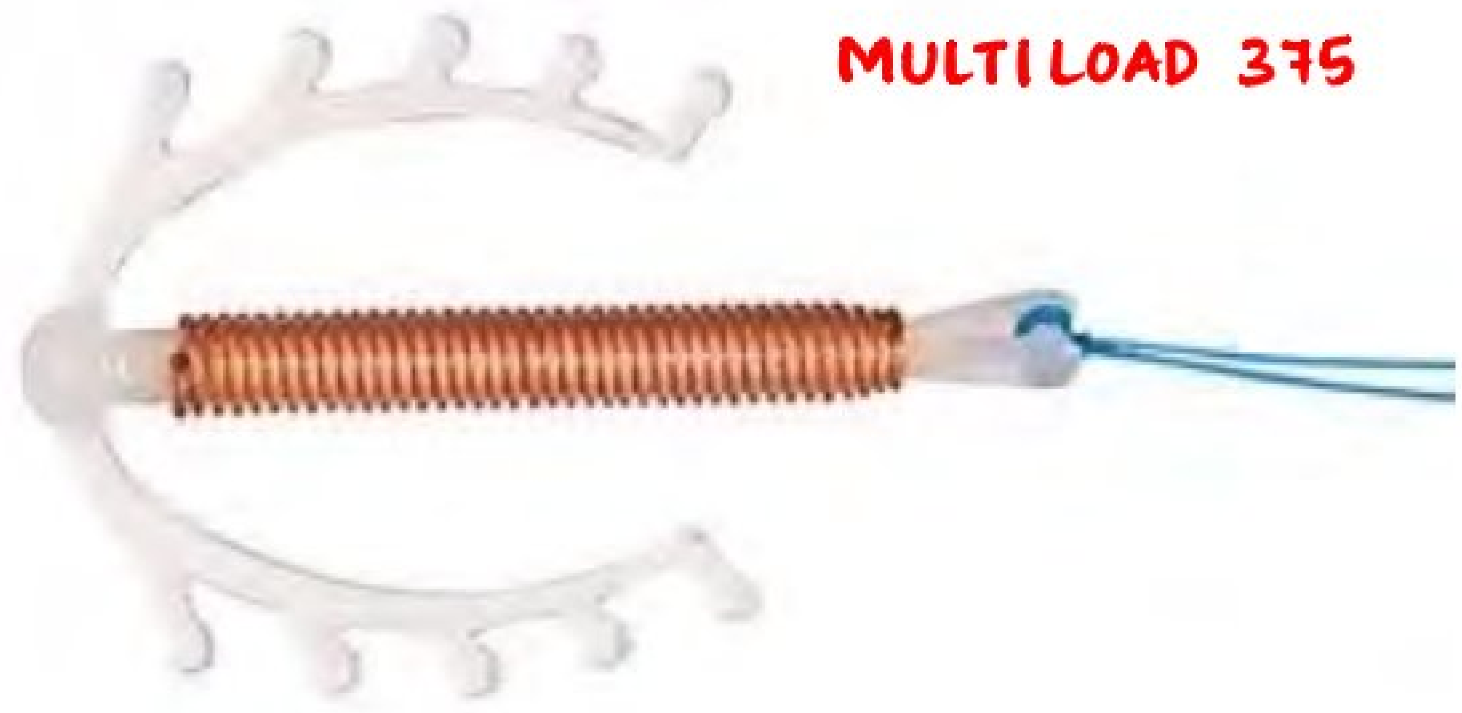
Ⓜⓒ organism : ACTINOMYCES

↓ Risk of PID because effect of LNG on Cervical mucus (THICK SCANTY)

LNG ⟶ Endometrium ⟶ ATROPHY

MULTILOAD 375

- Cu^{2+} device
- Arms Bent
- SA 375 : only on vertical limb
- Pre-loaded on INSERT
- Expulsion Rate less
- Life span - 5yrs



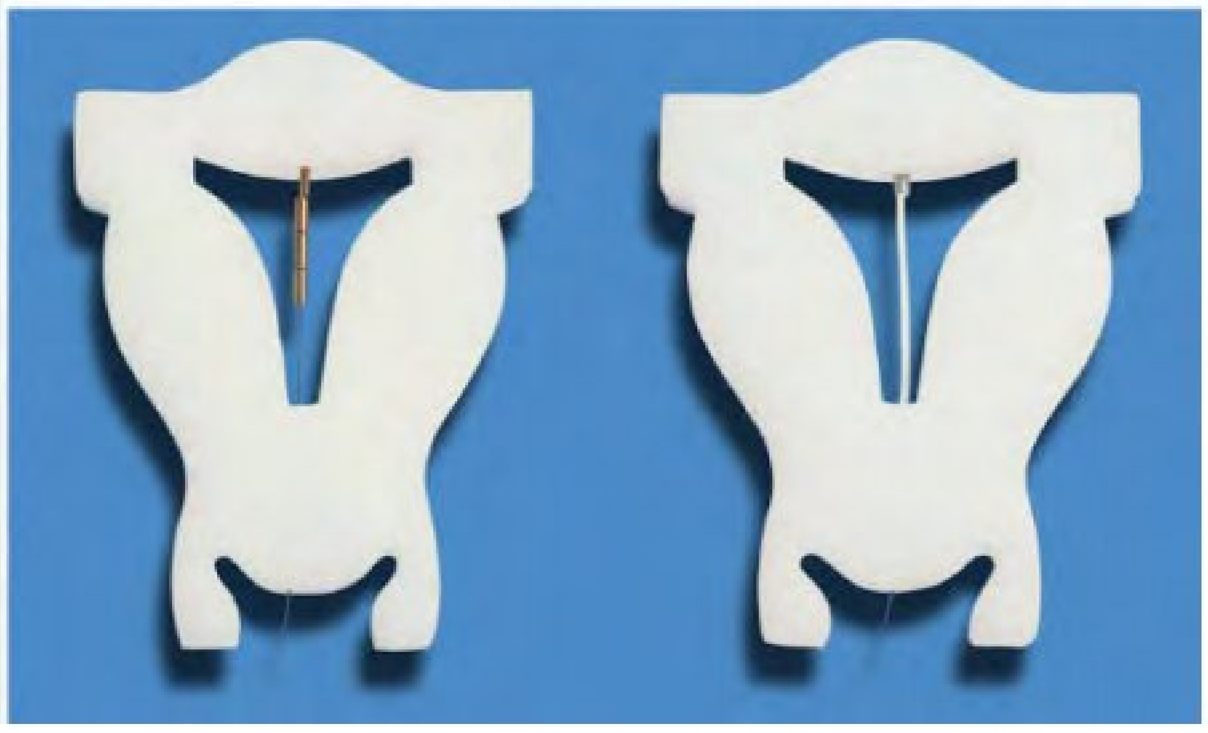
MULTILOAD 375

FRAMELESS IUD

- Pain is less ; good for Nulliparous ; 4th Gen

1. GYNEFIX 330 - copper cylinders - attached to Polypropylene suture
2. FIBROPLANT - LNG [14mcg/d , 20mcg/d]

FRAMELESS IUD



MEDICAL CONDITIONS AND CONTRACEPTION

- Diabetes and Heart failure → if not decompensated → IUD Safe even in HIV
  - No Need for Antibiotic Prophylaxis
  - +
  - Condom (Barrier) - prevents transmission.
- ↓ if decompensated
  - ↓ give Barrier method.

OCP

- Classified on Dose (Amount of Ⓔ) and Generations (Ⓟ)
- High dose - no longer used = ≥ 50mcg
- Low dose - < 50mcg (avg. 30-35 mcg)
- Very low dose - ≤ 20mcg
- FDA - 10mcg

Generations

1st Gen: NO Longer used for contraception eg. NORETHINDRONE

2nd Gen: LNG

3rd Gen: DESOGESTREL MC used

4th Gen: Spironolactone derivatives eg: DROSIPIRENONE

MALA N/D: Both have 0.03 mg of EE and 0.15mg of LNG + Ferrous fumarate (iron salts)
↳ free of cost

→ of LH surge
MOA of OCP: Inhibition of ovulation

Estrogen - inhibit folliculogenesis

Progesterone - acts on endometrium (mucus)

MC S/E: Breakthrough Bleeding

Ideal: Start 1st day of LMP
c̄in 5 days of LMP

if >5 days of LMP - use back up method contraception

- if misses 1 pill; it has to be taken as soon as possible/noticed. Next pill whenever its due
- if 2 pills are missed; back up method of contraception

F/R → Perfect use → 0.1
↳ Typical use → 8%

- fertility will return in most woman by 90 days

- Other drugs - ENZYME INDUCERS (↓ Efficacy of OCP) → Phenytoin/Phenobarb/Rifampicin
- Among Contraceptive method LEAST RISK OF ECTOPIC: OCP
- Reduce Risk of PID → If Infected → CHLAMYDIA, CANDIDA } SUBCLINICAL

- OCP's - ↓ Risk of Ovarian, Endometrial, Colon cancer but ↑ Risk of Cervical CA (if used >5yrs)
  Reversible

  CA BREAST - NO ↑ in Risk.
  Benign Breast ds - PROTECTIVE

  Hepatic Adenoma ↑

ABSOLUTE C/I

1. venous thromboembolism
2. CAD
3. Stroke
4. Migraine c̄ Aura
5. DM c̄ vasculopathy
6. K/c/o Breast CA
7. Age >35yr + Smoker
8. Suspected Pregnancy
9. Undiagnosed Bleeding
10. < 6wk Post partum
11. Derranged LIVER Enzymes
12. Breast feeding

MINI PILLS (POPs) - Very low dose Progesterone

- MOA : makes Cervical mucus - THICK
- should be taken at the same time everyday
- Safe Period - 3hrs
- Eg: CERAZETTE - 0.75mg DESOGESTREL ; only POP - MOA : inhibition of ovulation
  ↳ Safe period : 12hrs

MC S/E 1) Irregular Bleeding (METRORRHAGIA) 2) Cyst formation

CONTRACEPTION OF CHOICE

- in Breastfeeding patients - POP > IUD ; started immediately after delivery
- C/I : 1. Suspected Pregnancy 2. Current Breast CA 3. Undiagnosed Vaginal Bleeding

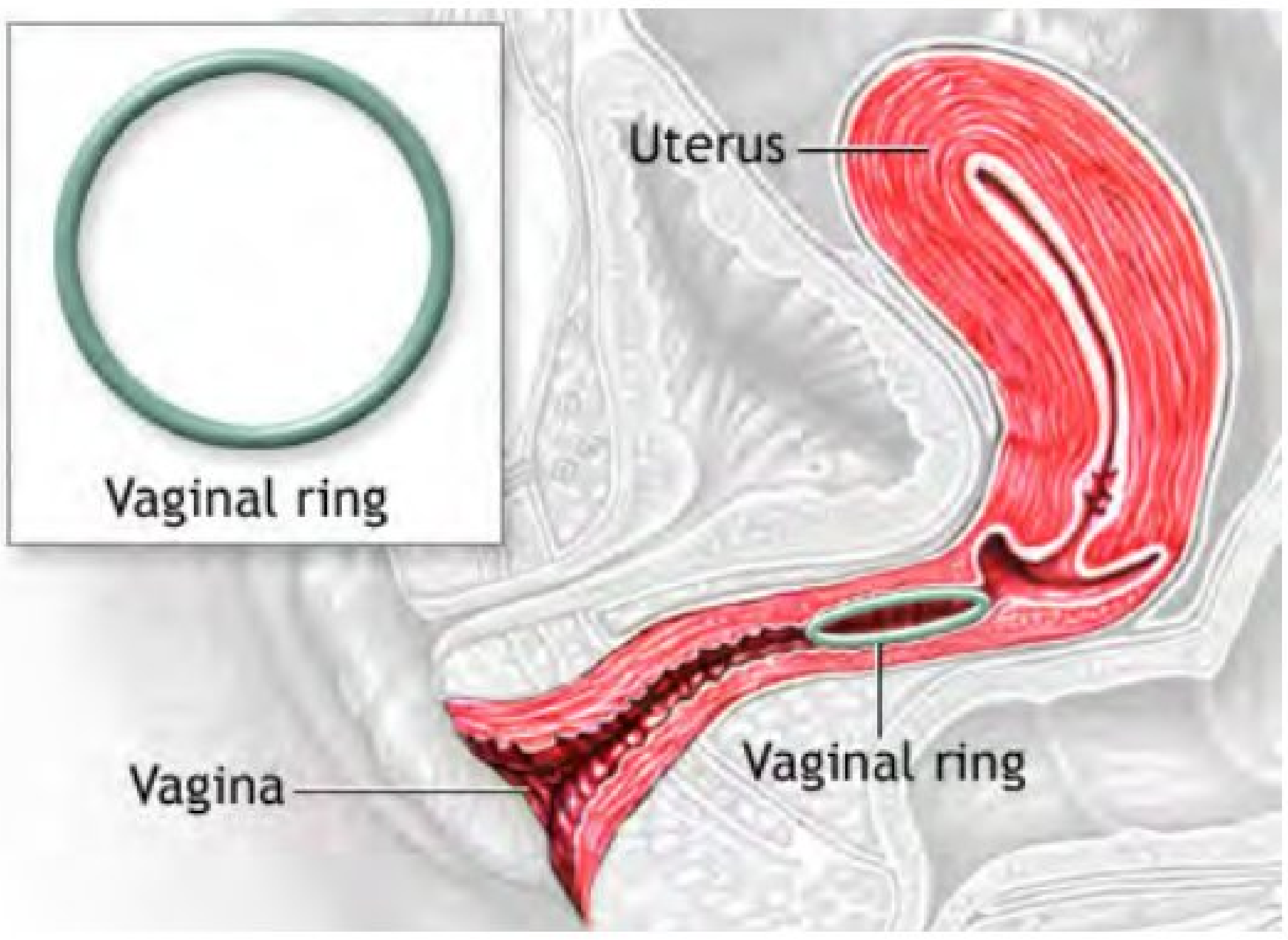
POP : ↑ Relative Risk of Ectopic

Previous h/o ECTOPIC - NOT A C/I

VAGINAL RING : NUVA Ring → E + P → ETONOGESTREL at 120mcg/d
E ↓ EE at 15mcg/d

MOA :
Absolute C/I :
Side Effects :
} same as OCP

- Outer dia = 55 mm
- Cross section = 4 mm
- Put in vagina 1st day of menstrual cycle ; remains in vagina for 3wks, removed after 1wk



- New Ring to be put after withdrawal Bleeding
- **Ideal :** Remain in vagina even at the coitus. If removed, then it should be put back ≤in 3hrs

TRANSDERMAL PATCH (E + P combo)

↗ Release Rate : 150 mcg/d

- 20 cm^2 patch ≤ 3 layers
- 0.75 EE + 6mg NORGESTINATE (3rd Gen P)

↳ Release Rate at 20 mcg/d

- New patch put every wk x 3wks ; then 1 wk patch free
- **MOA :** same as OCP
- **ABSOLUTE C/I :** same as OCP
- **↑ RISK OF VTE :** Patch >> OCP

NON-STEROIDAL CONTRACEPTION

1. SAHELI = Chaya (Free of Cost)

- CENTCHROMAN has ORMILOXIFENE (SERM)
- MOA : makes Endometrium Out of Phase (inhibits implantation)
- Developed at CDRI, Lucknow 1991
- S/E : Delay in Return of Menstrual cycle
- EACH TAB = 30mg, TWICE A WEEK 1st 3months then 1 pill/wk.

2. DMPA (DEPO MEDROXY PROGESTERONE ACETATE)

- Depo Provera
- given i.m (deltoid/Gluteus)
- FREE OF COST - ANTARA
- 150mg MPA
- Given every 3 months ; 4 inj. a year
- **MOA :** Inhibition of ovulation (cervical mucus - Thick, Endometrium - Atrophic)
- given ≤in 7days of LMP (≤in 24hrs)
- can be given in Breast feeding ; immediately after delivery
- ↓ PID Risk
- (MC) S/E Irregular Bleeding (Metrorrhagia) ; ↓ Bone density ; delay in Return of fertility (avg. about 12months)

- Absolute C/I : (P only compounds) • Undiagnosed Bleeding • K/c/o BREAST Cancer
  • Suspect Pregnancy
- Useful in pts of ① EPILEPSY - ↓ Seizure episode by ↑ seizure threshold
  ② SICKLE CELL ANEMIA - ↓ Sickling Crisis
- H/o ECTOPIC - NOT a C/I for DMPA

IMPLANT eg: IMPLANON ; SUBDERMAL

- Single ROD device
- Only Ⓟ → ETONOGESTREL 68mg at 67mcg/d
- 4cm long - Effective for Span of 3years
- have lowest F/R - 0.05% • Most Effective
- F/R of IMPLANON - 0.01%
- MOA : inhibition of ovulation ;
  acts on Cervical mucus, endometrium

Irregular Bleeding - (MC) S/E

- NO Delay in Return of Fertility
- 1st 7days after LMP • OPD Procedure
- ABSOLUTE C/I : same as other Ⓟ compounds

EMERGENCY CONTRACEPTION

- Most Effective EC : CuT ≤in 5days
- Most Effective EC : ULIPRISTAL > MIFEPRISTONE > LNG → (MC) used - 1.5mg tab SD or as 2 divided doses 12 hrs apart.
  - ULIPRISTAL ↙ • 30mg SD • SPRM
  - MIFEPRISTONE ↙ [RU 486] • 10-50mg SD • SPRM

OCP's can also be used.

MOA : delay in the ovulation by delay in LH surge > Inhibit Implantation

EC → Interceptives. Ideal EC : ≤in 72hrs of an unprotected intercourse

Effectivenes : all EC → upto 120hrs (5days)

• NO TEST Required • NO PELVIC Examⁿ Required

- Most effective at 5th day of unprotected intercourse = CuT > Ulipristal
- DANAZOL - 400mg BD ; 12hrs apart
- LNG - (MC) used EC ⟶ 0.75mg stat and after 12hrs
- EE 50mcg + LNG 0.25mg [YUZPE Method] - taken as 2 tab stat and 2 tabs after 12hrs

[Total = 200mcg + 1mg]

CONTRACEPTIVE PRESCRIPTION

1. Adolescent girls - Low dose COC pills
2. Newly Married Couple - OCP
3. Post Abortal : Pill > IUD
4. Post Partum
    - ↳ Lactating : POP > IUD (10wks)
    - ↳ if Non-Lactating : POP > IUD (3wks)
5. Interval - < 35yrs → PILL/IUD
    - > 35yrs → IUD
6. Stop future pregnancy - Sterilization
7. Older women - LOW DOSE pills can be continued till menopause
8. Women at Risk for STI's : Condom c̄ another method.

STERILIZATION

1. FEMALE STERILIZATION

- for PP - Minilap c̄ Modified POMEROY preferred ; done upto 7days PP (Post Partum)
- Laparoscopy used for interval sterilization - NOT ADVISABLE for PP pts for 6wks following delivery ; not in pt c̄ heart ds.
- Hb should not be < 8g/dl
- HIGHEST FAILURE RATE : CLIPS and BIPOLAR CAUTERY
- LEAST FAILURE RATE : UNIPOLAR CAUTERY and POST-PARTUM LIGATION
- Maximum chances of REVERSAL : Laparoscopic Tubal Ligation c̄ CLIPS
- Maximum Risk of ECTOPIC : BIPOLAR
- LEAST Risk of ECTOPIC : PP c̄ PARTIAL SALPINGECTOMY
- BEST PROGNOSIS for REVERSIBILITY - ISTHMOISTHMIC Type

VARIOUS TECHNIQUES FOR TUBAL STERILIZATION

A. IRVING PROCEDURE : least likely to fail

- Medial cut end → Buried in myometrium posteriorly
- Distal cut end → Buried in MESOSALPINX

B. POMEROY PROCEDURE

- a loop of oviduct ligated, knuckle of tube excised
- SIMPLEST
- F/R - 0.75%

C. MADLENER PROCEDURE

- knuckle of oviduct is crushed and then ligated s̄ out resection
- F/R : 7%

D. PARKLAND PROCEDURE: Modified POMEROY

- a mid segment of tube is separated from the mesosalpinx at an ovarian site and the separated tubal segment is ligated proximally and distally and then excised

F/R : 0.25%

E. KROENER PROCEDURE

- tube is ligated across the ampulla and distal portion of ampulla, including all of the fimbriae is resected.
- performed at any time, often done at C.S delivery

F. UCHIDA [NO FAILURE]

- Saline c̄ Epinephrine inj into subserosal area of tube
- Medial stump buried in MESOSALPINX
- Lateral stump ligated, kept outside MESOSALPINX - PURSE STRING SUTURE

ELECTROCOAGULATION TECHNIQUE

- causes greater tubal damage
- F/R → UNIPOLAR - 0.75%, BIPOLAR → 2.48%
- BIPOLAR CURRENT SAFER THAN UNIPOLAR ; but UNIPOLAR Coagulation appear to have the lowest failure rate.

MECHANICAL TECHNIQUE

1. FALOPE (YOON) RING Technique F/R - 1.77

- non-reactive silicone rubber band measure 3.6mm in Outer dia is used.
- loop of tube should clearly contain 2 complete lumens of tube.
- more painful POST OP d/t 2° ISCHEMIA

2. HULKA CLEMENS CLIP TECHNIQUE F/R - 3.65

- only 4mm of tube and virtually none of the blood supply is destroyed.

3. FILSHIE CLIP TECHNIQUE F/R - 0.9 - 1.2%

- clip of Titanium c̄ Silicone Rubber lining ; applied laparoscopically

INTERVAL TUBE FAILURE

1. Surgical errors - 30-50%

2. Pt was already pregnant at the time of Surgery - LUTEAL PHASE Pregnacy

3. Occlusion Method failure may be d/t fistula formation, which may follow Electrocautery procedures

4. Equipment Failure, s/a Defective Electric Current for the electrocautery also may be a causative factor

PUERPERAL STERILIZATION FAILURE

- **ECTOPIC PREGNANCY:** ½ of pregnancies that occur after a failed electrocoagulation procedure are ectopic

POST TUBAL LIGATION SYNDROME: 1. Pelvic discomfort 2. Ovarian cyst formation 3. Menorrhagia

MALE CONTRACEPTION

1. HORMONAL:
1. TESTOSTERONE ENANTHATE - 200mg wkly
2. TESTOSTERONE BUCICLATE - x 3-4 months
3. GnRH Antagonists c̄ TESTOSTERONE - Daily Injection.

2. GONADAL TOXINS:

PARTIAL REVERSAL:
1. GOSSYPOL ⟶ Hypokalemia
2. NITROFURANTOIN DERIVATIVES ⟶ Pulmonary fibrosis

SPERMICIDES:

1. BENZALKONIUM CHLORIDE : causes Electrolyte imbalance of aqueous phase of Cervical mucus coagulates ovulatory mucus

2. NONOXYNOL : has ANTI-CHLAMYDIAL Action ; INACTIVATED By SPERMACROSIN.

3. OCTOXYNOL : disrupt the cell membrane by interacting c̄ cell membrane lipoprotein

4. C31G 5. Polystyrene Sulphate

IMPLANTS

1. BIODEGRADABLE
1) Caproxor
2) Annuelle

2. NON-BIODEGRADABLE
1) NORMPLANT I and II
2) IMPLANON
3) UNIPLANT
4) ST-1435

INJECTABLES
1. DEPO PROVERA
2. MESIGYNA
3. CYCLOFEN
4. NET-EN
5. ORG 2154

MALE STERILIZATION

- Remove a piece of vas , atleast 1cm after clamping
- Not sterile upto 30 ejaculations ⟶ So, during that time use another contraception must be used for 3months
- **COST WISE : 5 VASECTOMIES to 1 TUBAL LIGATION**

Complications :

1. OPERATIVE : pain , scrotal hematoma , local infection (3%)

2. SPERM GRANULES : accumulation of sperm → presents 10-14day POST-OP. pain, swelling as hard 7mm large mass.

3. SPONTANEOUS RECANALIZATION - 0.6% f/u upto 3yrs

4. AUTOIMMUNE RESPONSE : 2% men have Abs against their own sperm

5. PSYCHOLOGICAL

(MC) cause : MISTAKEN IDENTIFICATION OF VAS.

GUIDELINES FOR STERILIZATION :

1. Age of male client below 60yrs

2. Age of female 22-49yrs

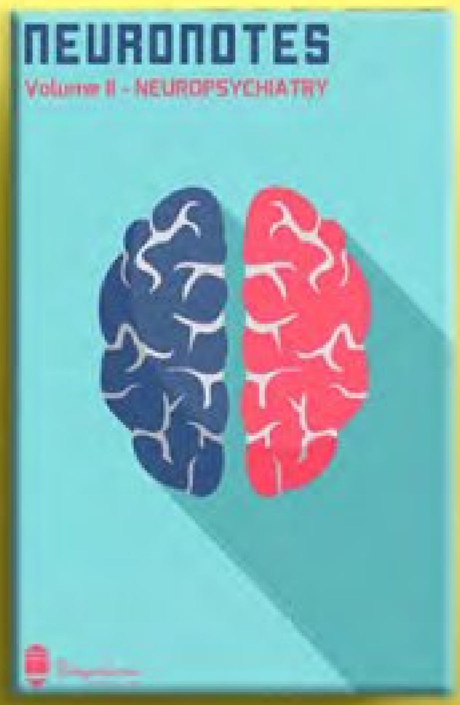
3. Client should be married or ever married

4. Couple must have 1 living child 1yr old at the time of the operation

5. If they have >2 children ; lower limit of husband / wife can be relaxed.

6. It is suffient if the acceptor declares having obtained consent of his/her spouse to undergo sterilization operation s̄out outside pressure , inducement or coercion.

Check out our awesome notes NEURONOTES



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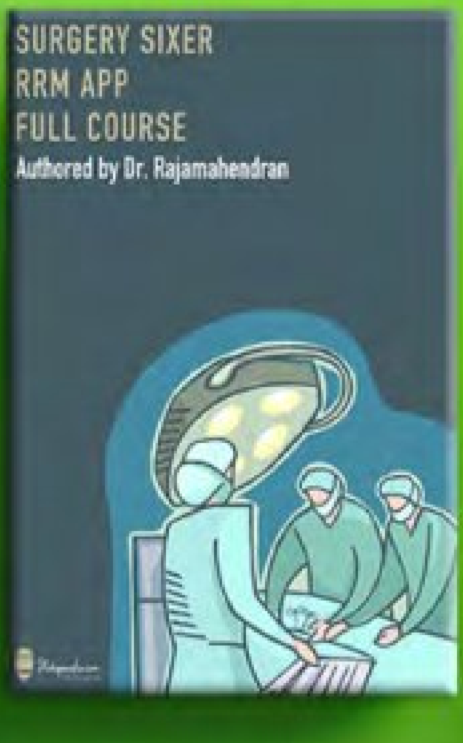

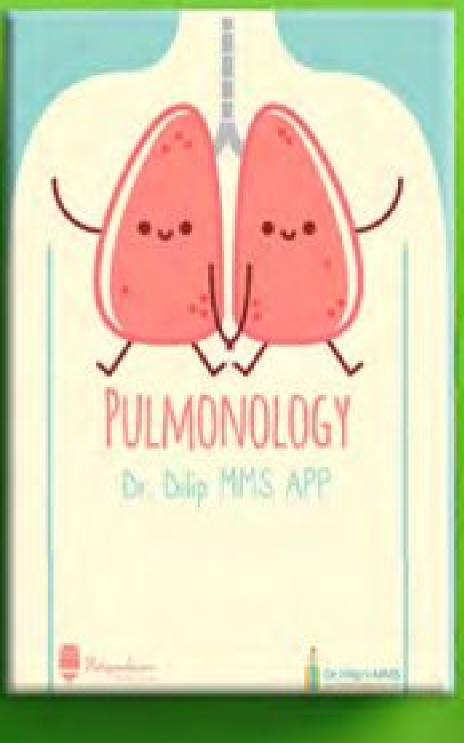

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